U.S. DEPARTMENT OF COMMERCE
National Technical Information Service

PB-278 171

Scientific and Technical Assessment Report on Vanadium

Health Effects Research Lab, Research Triangle Park, N C

Oct 77

PB 278 171

EPA-600/6-77-002
October 1977

STAR Series

SCIENTIFIC AND TECHNICAL ASSESSMENT REPORT ON VANADIUM

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U. S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

U.S. Environmental Protection Agency
Office of Research and Development
Washington, D.C. 20460

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO. EPA-600/6-77-002	2.	3. RECIPIENT'S ACCESSION NO. PB278171
4. TITLE AND SUBTITLE SCIENTIFIC AND TECHNICAL ASSESSMENT REPORT ON VANADIUM		5. REPORT DATE October 1977
		6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S)		8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS Health Effects Research Laboratory Office of Research and Development Environmental Protection Agency Research Triangle Park, NC 27711		10. PROGRAM ELEMENT NO. 1AA601
		11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS Health Effects Research Laboratory RTP,NC Office of Research and Development Environmental Protection Agency Research Triangle Park, N.C. 27711		13. TYPE OF REPORT AND PERIOD COVERED
		14. SPONSORING AGENCY CODE EPA-600/11

15. SUPPLEMENTARY NOTES

16. ABSTRACT

This report is a review and evaluation of the current knowledge of vanadium in the environment as related to possible deleterious effects on human health and welfare. Sources, distribution, measurement, and control technology are also considered. Vanadium is widely distributed in nature and is usually present in small quantities in all media and living forms.

The largest source of environmental contamination by vanadium is from oil combustion. Vanadium particulate matter is small, well within the respirable range for human beings. Respiratory absorption of vanadium appears to be very efficient, and it is thought that prolonged, repeated exposure could produce chronic lung disease. High-level industrial exposures of humans have shown evidence of complex internal injuries in the body, particularly enzyme interference.

The small particle size of vanadium emissions makes complete control unlikely, but good particulate control measures can greatly reduce vanadium output from most sources.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Vanadium	Health Effects	07B
Measurement	Industrial Exposure	06J
Control	Respiratory Absorption	06E
Toxicity	Biochemical Alterations	06F
Respiratory System	Environmental Appraisal	13B
		14B

18. DISTRIBUTION STATEMENT Release to Public	19. SECURITY CLASS (This Report) Unclassified	21. NO. OF PAGES 127
	20. SECURITY CLASS (This page) Unclassified	22. PRICE A07 - A01

EPA Form 2220-1 (9-73)

EPA-600/6-77-002
October 1977

SCIENTIFIC AND TECHNICAL ASSESSMENT REPORT ON VANADIUM

Program Element No. 1AA601

Assembled by
Health Effects Research Laboratory
Environmental Research Center
Research Triangle Park, North Carolina

for

U.S. ENVIRONMENTAL PROTECTION AGENCY
Office of Research and Development
Washington, D.C. 20460

RESEARCH REPORTING SERIES

Research reports of the Office of Research and Development, U.S. Environmental Protection Agency, have been grouped into series. These broad categories were established to facilitate further development and application of environmental technology. Elimination of traditional grouping was consciously planned to foster technology transfer and a maximum interface in related fields. These series are:

1. Environmental Health Effects Research
2. Environmental Protection Technology
3. Ecological Research
4. Environmental Monitoring
5. Socioeconomic Environmental Studies
6. Scientific and Technical Assessment Reports (STAR)
9. Miscellaneous Reports

This report has been assigned to the SCIENTIFIC AND TECHNICAL ASSESSMENT REPORTS (STAR) series. This series assesses the available scientific and technical knowledge on major pollutants that would be helpful in possible EPA regulatory decision-making regarding the pollutant or assesses the state of knowledge of a major area of completed study. The series endeavors to present an objective assessment of existing knowledge, pointing out the extent to which it is definitive, the validity of the data on which it is based, and uncertainties and gaps that may exist. Most of the reports will be multi-media in scope, focusing on single media only to the extent warranted by the distribution of environmental insult.

EPA REVIEW NOTICE

This report has been reviewed by the Office of Research and Development, EPA, and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

DISTRIBUTION STATEMENT

This report is available to the public from Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402.

Report No. EPA-600/6-77-002

PREFACE

Although this report is issued in the Scientific and Technical Assessment Report Series, it differs in several respects from the comprehensive multimedia format that publications in this Series usually have because it was nearly completed before the creation of the STAR series in August 1974.

This document was prepared by a task force convened under the direction of Dr. F. Gordon Hueter, Director, Special Studies Staff, U. S. Environmental Protection Agency, Environmental Research Center (ERC), Research Triangle Park (RTP), N. C. The Special Studies Staff assembled and produced the report. The objective was to review and evaluate the current knowledge of vanadium in the environment as related to possible deleterious effects on human health and welfare. Information from the literature and other sources has been considered generally through August 1973.

The primary reference for this report was a report prepared for the U. S. Environmental Protection Agency (EPA) by a National Academy of Sciences' Panel on Vanadium of the Committee on Medical and Biological Effects of Environmental Pollutants.

The following persons served on the ERC/RTP Task Force on Vanadium:

Robert Horton, Chairman
Marijon Bufalini
Gary Evans
J.H.B. Garner
Bruce Harris
James Homolya
Darryl von Lehmden
Sharon Long
Carol Sawicki
Jack Suggs
Elbert Tabor
Michael Waters
Douglas Worf

The substance of this document was reviewed by the National Air Quality Criteria Advisory Committee (NAQCAC) in public session on March 21, 1974. Members of NAQCAC were:

Arie J. Haagen-Smit, Chairman — California Institute of Technology
Mary O. Amdur — Harvard University
David M. Anderson — Bethlehem Steel Corporation
Anna M. Baetjer — Johns Hopkins University
Thomas D. Crocker — University of California
Samuel S. Epstein — Case Western Reserve University
C. C. Li — University of Pittsburgh

James McCarroll — University of Washington
Eugene P. Odum — University of Georgia
Elmer Robinson — Washington State University
Morton Sterling — Wayne County Michigan Health Department
Arthur C. Stern — University of North Carolina
Elmer P. Wheeler — Monsanto Company
John T. Wilson, Jr. — Howard University
Ernst Linde, Executive Secretary

The report was reviewed, under the supervision of Kenneth Cantor, by a task force composed of members of EPA's Office of Research and Development.

Review copies of this document were also provided to other government agencies and to industrial and public interest groups.

All comments and criticisms were reviewed and incorporated in the document where deemed appropriate.

TABLE OF CONTENTS

Page

LIST OF FIGURES vi

LIST OF TABLES vii

LIST OF ABBREVIATIONS AND SYMBOLS ix

1. INTRODUCTION 1-1

2. SUMMARY AND CONCLUSIONS 2-1
   - 2.1 SUMMARY 2-1
   - 2.2 CONCLUSIONS 2-4

3. CHEMICAL AND PHYSICAL PROPERTIES 3-1
   - 3.1 REFERENCE FOR SECTION 3 3-2

4. SAMPLING, PREPARATION, AND ANALYSIS 4-1
   - 4.1 INTRODUCTION 4-1
   - 4.2 ENVIRONMENTAL SAMPLES 4-3
   - 4.3 BIOLOGICAL MATERIALS 4-12
   - 4.4 STATIONARY SOURCE SAMPLES 4-14
   - 4.5 REFERENCES FOR SECTION 4 4-17

5. ENVIRONMENTAL APPRAISAL 5-1
   - 5.1 OCCURRENCE 5-1
   - 5.2 CONCENTRATIONS 5-9
   - 5.3 TRANSPORT AND MODELING 5-35
   - 5.4 REFERENCES FOR SECTION 5 5-41

6. EFFECTS OF VANADIUM 6-1
   - 6.1 BIOMEDICAL EFFECTS 6-1
   - 6.2 EFFECTS IN PLANTS AND MIROORGANISMS 6-23
   - 6.3 EFFECTS IN ANIMALS 6-26
   - 6.4 EFFECTS ON MATERIALS AND THE ENVIRONMENT 6-27
   - 6.5 REFERENCES FOR SECTION 6 6-30

7. CONTROL TECHNOLOGY 7-1
   - 7.1 REFERENCES FOR SECTION 7 7-2

LIST OF FIGURES

Figure — *Page*

5.1 Histogram for NASN Urban Sites, 1965-1969 5-15

5.2 Histogram for NASN Nonurban Sites, 1965-1969 5-16

5.3 Geographic Distribution of Vanadium Concentrations. Generalized on Basis of 5-year (1965-1969) Averages for NASN Sites 5-17

5.4 Location of Fixed Sampling Stations in Kanawha River Valley. Average Vanadium Concentrations (ng/m^3) for the Study Period (1964-1965) Indicated for Selected Sites 5-20

5.5 Vanadium Concentrations for Chicago (24-hour averages, in ng/m^3) 5-21

5.6 Monthly Variation of Vanadium Deposition, Helsinki, Finland, 1964-1965 5-25

5.7 Daily Variation of Total Particulate and Vanadium Concentrations in Air of Oil-heating Area during Cold Period, Helsinki, Finland, 1964–1965 5-26

5.8 Biogeochemical Vanadium Cycle 5-39

5.9 Flow Diagram of Fate of Vanadium in an Urban Air Shed 5-40

6.1 Chemical Composition (Minor Elements) of Harbor Sediment and Fly Ash as Compared with Sewage Sludges and Various Sediments and Soils 6-29

6.2 Temporal Variations of Lead, Cadmium, Uranium, and Vanadium Concentrations in Norwegian Glacier Ice 6-30

LIST OF TABLES

Table		*Page*
3.1	Some Physical Properties of Important Vanadium Compounds	3-1
3.2	Boiling Points of Vanadium Halides and Oxyhalides	3-2
4.1	Colorimetric Reagents for Detecting Vanadium	4-6
4.2	Analytical Results for "Standard" Rocks	4-12
5.1	Emissions of Vanadium by Source, 1968	5-2
5.2	Material Balance for Vanadium at a Coal-Fired Steam Power Plant	5-3
5.3	Vanadium Concentration of Coal Fly Ash, by Particle Size	5-3
5.4	Vanadium Concentration in Gasoline, Fuel Additives, Motor Oil, Fuel Oil, Crude Oil, and Coal	5-4
5.5	Atmospheric Concentrations of Vanadium at Boundary of Typical Steel Plant, 1967	5-5
5.6	Steels Containing Vanadium	5-6
5.7	Metallurgical Uses of Vanadium in the United States	5-6
5.8	Concentrations of Vanadium Pentoxide in Air at Point Sources at a Steel Plant	5-8
5.9	Annual Average Vanadium Concentrations — NASN Urban Sites, 1965-1969	5-11
5.10	Annual Average Vanadium Concentrations — NASN Nonurban Sites, 1965-1969	5-12
5.11	NASN Urban Sites Exceeding 100 ng/m^3 and Nonurban Sites Exceeding 10 ng/m^3, 1968	5-13
5.12	Vanadium Concentrations by Season, 1968	5-14
5.13	Quarterly and Annual Size Distribution for Vanadium, 1970	5-18
5.14	Vanadium Concentrations, Kanawha Valley Study	5-19
5.15	Seasonal Ambient Vanadium Concentrations — Birmingham, Alabama Area, 1964-1965	5-20
5.16	Annual Average Vanadium Concentrations, New York City, 1968	5-22
5.17	Annual Average Vanadium Concentrations, Helsinki, Finland, 1962-1963	5-22
5.18	Monthly Vanadium Deposition, Helsinki, Finland, 1964-1965	5-23
5.19	Vanadium Deposition at Various Investigation Points, Helsinki, Finland, 1964-1965	5-23
5.20	Average Vanadium Concentrations, December to February 1965, Helsinki, Finland	5-24
5.21	Vanadium Concentrations in Food	5-28
5.22	Vanadium Concentrations in Animal Specimens	5-29
5.23	Vanadium Concentrations in Fruits and Vegetables	5-30
5.24	Vanadium Concentrations in Fats and Oils	5-31
5.25	Vanadium Concentrations in Plants and Animals	5-32
5.26	Vanadium Concentrations in Tissues of Wild Animals	5-33
5.27	Vanadium Concentrations in Human Tissues	5-36
5.28	Estimated Particle Size Distribution (weight percent)	5-40
6.1	Balance of Vanadium in Two Men	6-2
6.2	Vanadium Intake in Urban and Nonurban Areas	6-2

6.3 Excretion of Vanadium by Three Human Subjects 6-5
6.4 Thermal-Precipitator Samples Taken from Superheater during Cleaning Operation 6-8
6.5 Analysis of Dust from Boiler during Cleaning 6-8
6.6 Symptoms in Vanadium Workers 6-9
6.7 Physical Findings in Vanadium Workers 6-9
6.8 Threshold Limit Values for Vanadium Compounds, 1961, 1971, 1972 6-10
6.9 Documentation of Threshold Limit Values: Outline of Literature Findings Cited on Industrial Exposures to Vanadium 6-11
6.10 Comparison of 1972 TLV for Vanadium Pentoxide Dust and Fume with TLVs for Other Metallic Oxides 6-12
6.11 Toxicity of Vanadium Salts to Mammals 6-15
6.12 Lethal Doses of Selected Vanadium Salts 6-16
6.13 Respiratory Effects of Vanadium Pentoxide in Experimental Animals 6-17
6.14 Vanadium in Ash of Above-Ground Parts of Plants Grown in Experimental Plots 6-25
6.15 Comparison of Vanadium in Ash of Tops and Roots of Plants Grown in Experimental Plots 6-25

LIST OF ABBREVIATIONS AND SYMBOLS

ADP	Adenosine diphosphate
ATP	Adenosine triphosphate
ASV	Anodic stripping voltammetry
BOF	Basic oxygen furnace
b.p.	Boiling point
°C	Degrees Celsius (centigrade)
$CaCO_3$	Calcium carbonate
$Ca_3(PO_4)_2$	Calcium phosphate
$CaSO_4$	Calcium sulfate
$Ca(UO_2)_2(VO_4)_2.nH_2O$	The mineral tyuyamunite
CHESS	Community Health and Environmental Surveillance System
cm	Centimeter
EPA	U. S. Environmental Protection Agency
ESP	Electrostatic precipitator
Fe_2O_3	Ferric oxide
g	Gram
Ge(Li)	Lithium-drifted germanium detector
Hg	Mercury
HSLA	High-strength, low-alloy steel
kg	Kilogram
km	Kilometer
$K_2(UO_2)_2(VO_4)_2.3H_2O$	The mineral carnotite
lb	Pound
LTA	Low-temperature asher
m	Meter
m^3	Cubic meter
Mg	Megagram (metric ton, MT)
mg	Milligram
MIBK	Methyl isobutyl ketone
min	Minute
mm	Millimeter
MMD	Mass median diameter
MT	Metric ton (megagram, Mg)
NaI(T1)	Thallium-activated sodium iodide detector
Na_2O	Sodium oxide
NASN	National Air Sampling Networks
ERC/RTP	EPA Environmental Research Center, Research Triangle Park, North Carolina
ng	Nanogram
ppb	Parts per billion
ppm	Parts per million
psi	Pounds per square inch
Si(Li)	Lithium-drifted silicon detector
TLV	Threshold limit value
V	Vanadium

VCl_3	**Vanadium trichloride**
$VOCl_2$	**Vanadyl dichloride**
$VOCl_3$	**Vanadyl trichloride**
V_2O_3	**Vanadium trioxide**
V_2O_4	**Vanadium tetroxide**
V_2O_5	**Vanadium pentoxide**
yr	**Year**
μg	**Microgram**
μm	**Micrometer (micron)**

ABSTRACT

This report is a review and evaluation of the current knowledge of vanadium in the environment as related to possible deleterious effects on human health and welfare. Sources, distribution, measurement, and control technology are also considered. Vanadium is widely distributed in nature and is usually present in small quantities in all media and living forms. The concentration of vanadium in ambient air varies a great deal across the United States, with the highest levels — annual averages exceeding 100 ng/m^3 — occurring in metropolitan areas on the eastern seaboard.

The largest source of environmental contamination by vanadium is oil combustion. Vanadium particulate matter is small, well within the respirable range for human beings. Most information on human vanadium exposure is of occupational origin, principally exposure to dusts in metallurgical work and boiler-cleaning operations. Respiratory absorption of vanadium appears to be very efficient, and it is thought that prolonged, repeated exposure could produce chronic lung disease. High-level industrial exposures of humans have shown evidence of complex internal injuries in the body, particularly enzyme interference.

The small particle size of vanadium emissions makes complete control unlikely, but good particulate control measures can greatly reduce vanadium output from most sources.

SCIENTIFIC AND TECHNICAL ASSESSMENT REPORT ON VANADIUM

1. INTRODUCTION

The purposes of this document are to explore current knowledge regarding the sources and distribution of vanadium in the environment, to study its presence and role in living things, and to evaluate its effects on human health and welfare. Particular attention is given to the air environment, its pollution, and undesirable effects of such pollution. Interest in vanadium as a pollutant, particularly in air, originates from its capacity to produce severe respiratory illness in workers exposed to vanadium compound dusts and fumes. Human and experimental animal observations also indicate the presence of metabolic changes in other organs resulting from vanadium overexposure, but these are not as well understood as the respiratory effects.

2. SUMMARY AND CONCLUSIONS

2.1 SUMMARY

Vanadium occurs commonly but not uniformly in the earth's crust. The average concentration is about 150 milligrams per kilogram (mg/kg). It is extracted from ores usually containing 0.1 to 1.0 percent of vanadium compounds, with a maximum of 3.0 percent. It is frequently associated in ores with uranium and titanium, and it is mined in conjunction with these metals. Vanadium is also widely distributed in small amounts in living things. High concentrations are rare in nature, but are sometimes produced by human activities.

Current measurement methods for vanadium are more sensitive and reliable than older methods, which often produced erroneous data. Environmental and biologic information is still quite sparse for this metal. No comprehensive comparative studies of analytical methods have yet been made; thus no recommendations regarding a best method can be made at this time.

By far the largest source of environmental contamination with vanadium from human activity in this country is from the burning of oil and, to a lesser extent, of coal. This pollution results mainly from emissions into the air of very fine particles, but the ash from combustion of both types of fossil fuel also contains the metal. Disposal of this ash may pollute soil or water. The vanadium content of coal is moderately variable, but oils differ more widely. Venezuelan oil imported into the northeastern part of the country has high vanadium content; other oils used in the United States are quite low in vanadium. Refining processes virtually eliminate vanadium in kerosene, gasoline, diesel fuel, and home heating oils. Vanadium is concentrated in the residual oils that are burned (along with crude oil) in large heating and power units, petroleum refineries, and ships. Desulfurization of oil removes vanadium in quantities proportional to the amount of removed sulfur.

Extraction and use of vanadium also produce some opportunities for environmental contamination. Extraction of the metal from ores begins with sintering, which emits fine particulates to the air. Extracting is followed by leaching with water, which may produce contamination of soil or water. Initial alloying into ferrovanadium in the few ferrovanadium plants in this country produces fume in large quantities, which may produce air pollution. Use of the vanadium metal and compounds (particularly the latter) is widespread. The iron and steel industry is the largest user of vanadium. The chemical industry commonly uses vanadium catalysts, and there are some uses for vanadium in the ceramic, dye, and ink industries — nearly all of which use very small quantities, which are then widely dispersed. Opportunities for environmental contamination from use sources are presently thought to be limited to recycling (for steel alloys) and disposal of spent catalysts and other products. The latter groups present many uncertainties. In steel reclamation in basic oxygen furnaces, some of the alloying substances are driven off in fume. Local contamination may result, depending on the extent of particulate control used.

The concentration of vanadium in ambient air varies a great deal across the United States, with the highest levels — annual averages exceeding 100 nanograms per cubic meter (ng/m^3) — occurring in a cluster of cities along the eastern seaboard. The primary source of airborne vanadium in that area is the large quantity of fuel oil (much of which comes from the vanadium-rich crude oil of Venezuela) used for generation of electricity and for space heating.

In spite of wide fluctuations from year to year, vanadium levels have been generally increasing in recent years, probably in response to the increasing use of oil as a fuel. Winter averages are more than triple summer averages, reflecting the seasonal pattern in fuel oil consumption for heating purposes.

Studies of particle size distribution have shown that most vanadium-bearing particulate matter is very small — well within the respirable range for human beings.

A number of intensive air pollution studies in metropolitan areas have confirmed that maximum vanadium concentrations occur in areas of greatest population density during the coldest part of the year and during the late evening hours.

Vanadium soil concentrations vary according to the type of rock or soil and are highest in shales and clays. The estimated average concentration in the earth's crust is 150 mg/kg. The concentration of vanadium in fresh water varies with the soil and rocks in the area of the water source being sampled. Because of the presence of vanadium-containing uranium ores in the rocks, the rivers of the Colorado Plateau have the highest vanadium levels. Vanadium concentrations in fresh water vary from below detectable to 300 micrograms per liter (μg/liter). Vanadium entering the ocean from fresh water sources in usually deposited in sediments. Only about 0.001 percent of the vanadium entering the oceans remains in soluble forms. The total amount of vanadium in the oceans is estimated to be 7.5 x 10^{12} kg. The concentration in sea water ranges from 0.2 to 29 μg/liter.

Vanadium is ubiquitous at low levels in foods as far as is now known. However, data are meager and unreliable because of the older, poorly developed methodology of some studies.

All plants contain vanadium. Concentrations of the element tend to be higher and related to soil levels of vanadium in roots, and lower and independent of soil levels in leaves. The metal is essential for production of soil and plant nitrate by the bacteria of the root nodules of legumes.

Small amounts of vanadium are present in most animal tissues. Levels are somewhat higher in fat and in keratin-containing tissues such as hair, nails, and hooves. Accumulation with age in human lungs has been noted, but this effect has not been seen in other animals. Data on this subject are limited.

Information for modeling the movement of vanadium in the environment is grossly inadequate. This applies even to air, where the largest body of data exists.

Most information on human vanadium exposure is of occupational origin. Vanadium exposure in industry is principally from dusts in metallurgical work and boiler-cleaning operations. Nonindustrial exposure is from foodstuffs, with small amounts coming from water and air. Gastrointestinal absorption of vanadium is poor, but respiratory absorption may be very efficient. The percentage of vanadium that enters the body through the lungs is high as compared to other metals, with vanadium from contaminated air apparently accumulating in the lungs with age. Absorbed vanadium is transported in serum lipid fractions to the highly vascular organs and is excreted in the urine and to a lesser extent in the feces. Retained vanadium is stored mainly in fat. Keratin may be a minor storage depot.

Oxides and salts of vanadium are respiratory irritants at levels lower than those resulting in systemic toxicity. Clinical symptoms of acute exposure to vanadium pentoxide aerosol at 0.1 to several mg/m^3 consist of inflammation of eyes and throat, persistent cough, and tightness of the chest. The onset of symptoms is often delayed by several hours, depending on the severity of the exposure; however, symptoms may persist for a week or more. "Green tongue" results from the oral reduction of the pentoxide to the trioxide. Some individuals may become sensitized to vanadium so that they exhibit dermal and more severe respiratory symptoms on subsequent exposures at the same concentrations.

Chronic inhalation of vanadium pentoxide dusts in industry has resulted in inflammation of the respiratory tract, chronic productive cough, wheezing, shortness of breath, and fatigue. Pneumonitis and bronchopneumonia have also been observed. Vanadium workers are probably predisposed to secondary respiratory infections, which may account for certain chronic changes in respiratory tract tissues. Exposure to vanadium may be detected by elevated vanadium levels in urine and depressed cystine content of fingernails.

It is unlikely that the positive correlations between vanadium content in urban air and mortality from various causes in two exploratory statistical studies reflect a causal relationship. Excess mortality for causes examined in these studies has not been reported in vanadium workers, who are exposed to higher levels of the metal and its compounds than are community residents. In addition, the studies were incompletely adjusted for important, probably relevant factors.

Vanadium salts may reduce serum cholesterol levels in young men and animals, and they are not very toxic when given orally. Fifty to 200 mg/day for many weeks have been administered without production of symptoms. Vanadium salts are highly toxic when given intravenously; lethal doses range from 1 to 20 mg/kg in lower animals and man.

The irritative respiratory effects of vanadium oxides and salts in experimental animals (rats, mice, and rabbits) are, in general, similar to those reported in man. However, the higher concentrations employed in acute animal exposures have produced marked vascular injury in the lungs and in internal organs as well. It is believed that absorption of vanadium in severe acute exposure (tens of mg/m^3) is responsible for damage to the liver, kidney, heart, and brain by causing vascular constriction, congestion, and hemorrhage. Intermittent exposure (for 1 to 2 hours daily or every other day) at lower levels (3 to 5 mg/m^3 vanadium pentoxide as condensation aerosol) produces hemorrhagic inflammation only in the lungs. In one study, however, continuous exposure at very low levels (0.005

to 0.027 mg V_2O_5 fume/m^3) produced hemorrhagic respiratory inflammation as well as vascular congestion and hemorrhage in other internal organs.

Biochemical alterations resulting from vanadium exposure include depression of synthesis or increased destruction of cystine and cysteine with an overall lowering of serum protein sulfhydryl groups. Serum levels of ascorbic acid are lowered (perhaps because of insufficient cysteine as gluthathione to maintain the oxidized state) such that ascorbate no longer facilitates removal of iron from ferritin. Hemoglobin synthesis is probably, therefore, reduced, and the lowered hemoglobin levels and anemia that accompany severe chronic exposure to vanadium are manifested clinically.

The reduced hepatic levels of coenzyme A following exposure to vanadium may reflect the reduced availability of cysteine-a precursor in the biosynthesis of coenzyme A. Coenzyme A, in turn, plays a central role in many biosynthetic and oxidative pathways. Several other enzyme disturbances have also been observed in animal studies. Continued investigation may be expected to elucidate the interrelated biochemical and physiological effects and cellular damage resulting from vanadium exposure. Whether a mutagenic, carcinogenic, or teratogenic potential exists for vanadium remains to be determined.

Vanadium has been shown to be an essential nutrient for chicks and rats. It is essential for soil nitrogen-fixing microorganisms. The nutritional requirement for vanadium has not been widely studied. It is probable that vanadium is essential to many plants and animals, but its specific role has not been determined. Spontaneous deficiency states have not been observed.

Vanadium has been found to accumulate to very high levels in sea squirts and in agaric mushrooms, but these are rarely eaten. No other examples of this phenomenon are known. No reports of plant damage from vanadium have been located. Only one episode of damage to animals was reported, and this was caused by contamination of a cow pasture with soot that had been removed from a nearby oil-fired boiler. No information has been found on damage to materials from airborne vanadium. However, vanadium in fuels, particularly residual fuel oils, is very corrosive to boilers and piping. This effect is apparently due to direct action and also to catalytic oxidation of sulfur dioxide to sulfuric acid. Fuel additives are employed to counteract this damage, but their mechanism and effect on emissions are not well understood.

Standard fine particulate control techniques, when properly applied, accomplish good control of vanadium emissions. These techniques are not at present widely applied to oil combustion or ferrovanadium production. Because the aerosol particle size is characteristically very small, high collection efficiency is important.

2.2 CONCLUSIONS

Vanadium is widely distributed in nature in the physical and biological environment. It is usually present in small or very small quantities in all media and living forms.

Moderate quantities of vanadium are extracted for industrial use. Present uses are in small to very small amounts in widely distributed products, mainly metal alloys.

Vanadium is present in significant amounts in coal and oil. Some oils, particularly those from Venezuela, have a high vanadium content. Refining produces light fuels — for example, gasoline, kerosene, and diesel oil — that are virtually free of vanadium. The vanadium is concentrated in the residual fractions used for large heating and power plants. Desulfurization of oil reduces the vanadium proportionally to the sulfur reduction.

About 17,000 megagrams (metric tons) of vanadium are emitted to the atmosphere of this country each year. Of this, 88 percent comes from oil, 9 percent from coal, and the remainder from industrial sources. Owing to the uneven distribution of high-vanadium-content oils and to seasonal heating requirements, emissions resulting from oil combustion are higher in the northeastern United States and in the first and last quarters of the year.

Some of the sources and distributions mentioned provide opportunity for known or possible sources of overexposure. The natural distribution is thought to be innocuous. Unless proper precautions are used, workers in ore extraction, ferrovanadium production, and the cleaning of oil ash from boilers suffer severe injury to the eyes and the respiratory system. Local public exposures are possible in the vicinity of extractive plants, ferrovanadium plants, and steel recovery plants. The extent of environmental contamination in the neighborhood of such sources operating under current conditions is not known. No reports of neighborhood injury have been found, but no careful studies have been made. The fuel consumption sources produce lower community exposure levels over much more extensive areas. These are, of course, highest in large cities that have multiple large sources. Epidemiologic studies for possible injury in these circumstances are meager and inconclusive. Disposal of vanadium-containing products and wastes — for example, slags, ashes, and spent catalysts — has been poorly evaluated for possible problems. As noted earlier, incidence of cattle poisoning from improper disposal of soot has been described. Contamination of air, water, and land may also occur from these sources.

Good particulate control measures can greatly reduce vanadium output from most sources. The very small particle size characteristic of vanadium emissions makes complete control unlikely. Oil combustion emissions are not ordinarily controlled. Particulate emissions from the other sources, including incineration, are being increasingly controlled. If desulfurization continues in use or is increased, it will decrease emissions further. The high vanadium content of spent catalyst, coal ash, and oil ash (particularly the latter) as compared to low content in ores, has produced some interest in recycling. If reclamation develops, it would reduce the danger inherent in unsupervised disposal.

Experimental human exposure to 0.1 mg/m^3 vanadium pentoxide dust for 8 hours produced mild excess mucus production and cough beginning the following day and lasting 8 days. No general symptoms, fever, alteration of pulmonary function, or disability were present. Higher levels of exposure produced more severe and longer lasting effects. Most or all of the effects produced are reversible, but recovery from high-level exposure may take weeks or months. Prolonged repeated exposure is thought to produce irreversible chronic lung disease and promote infection in the lower respiratory tract. No well-designed studies have been carried out to confirm this clinical impression. Long-term air vanadium levels reported by the National Air Sampling Networks (NASN) do not exceed 1 μg/m^3, and most are far below this level. The maximum individual concentration recorded is 2.5 μg/m^3 for 24 hours, which is far below the lowest experimental dose described above. Information on the extent of and effects from human exposure to air vanadium concentrations between 1 and 100 μg/m^3 is needed to determine critical exposure levels. Laboratory exposure experiments, industrial data, and field studies would all be helpful.

High-level industrial exposures of men and experimental exposure of animals, unlike those described above, have shown evidence of complex internal injury in the body, particularly interference with a number of enzymes, in addition to the local respiratory tract irritation. These phenomena have not been adequately evaluated at lower exposure levels, nor are their interrelationships and injury potential understood. Intestinal and digestive disturbances resulting from vanadium have not been observed, possibly because of the very low rate of absorption.

The possibility of carcinogenesis, mutagenesis, or teratogenesis as a result of vanadium has not been investigated.

Vanadium is known to be essential to rats, chicks, and nitrogen-fixing bacteria, and it may be essential to many other living forms as well. Spontaneous deficiencies have not been reported. Although there are no available data showing essentiality in man, animal data indicate that vanadium is probably an essential element in human nutrition.

Vanadium injury to animals or plants is rare or unknown. When present in fuel oils, it produces considerable damage to boilers and other metal equipment.

Available data indicate in general that vanadium in the concentrations found in air, food, and water is not a health hazard. A significant research effort to evaluate vanadium distribution in the environment and its potential threat to health therefore does not appear to be warranted at this time. Particular potential problem areas, however, should be monitored for environmental vanadium, and the health status of persons residing or working in such areas should also be followed if evidence of environmental contamination appears.

3. CHEMICAL AND PHYSICAL PROPERTIES

Pure vanadium (V) is a bright white, soft, ductile metal having the following properties: atomic weight 50.942, atomic number 23, melting point 1890° ± 10 degrees Celsius (°C), boiling point 3380°C at 760 millimeters of mercury (mm Hg), specific gravity 6.11 at 18.7°C, and valence 0, 2+, 3+, 4+, or 5+. Placed in a simple aerated saline solution, vanadium has a high resistance to corrosion — at least as high as stainless steel and probably as high as titanium. Vanadium is inferior to stainless steel in its resistance to corrosion in concentrated alkaline solutions, but resistance is good in dilute solutions of alkalies, sulfuric acid, and hydrochloric acid. Pure vanadium resists oxidation up to about 690°C, but the oxidation rate of the metal is very rapid above that temperature. The melting point of vanadium is at least 400°C higher than the melting point of most steels. Some physical properties of important vanadium compounds are listed in Table 3.1.

Table 3.1. SOME PHYSICAL PROPERTIES OF IMPORTANT VANADIUM COMPOUNDS[1]

Compound	Melting point, °C	Boiling point, °C	Solubility in water, g/100 cm^3	
			Cold	Hot
Vanadium pentoxide	690	1750	0.8	No data
Vanadium trioxide	1970	No data	Slightly soluble	Soluble
Sodium metavanadate	630	No data	21.1	38.8
Vanadium tetrachloride	28 ± 2	148.5	Decomposes	No data
Vanadium oxychloride	No data	126.7	Decomposes	No data
Ammonium vanadate	200[a]	No data	0.52	6.95[a]

[a] Decomposes.

Vanadium exists in a number of valence states in a large variety of compounds. In general, the halides are hygroscopic and hydrolyze in water. Both the halides and oxyhalides are volatile — as indicated by their low boiling point (Table 3.2). Several oxides exist; V_2O_5 is sold commercially and is formed when the vanadium oxides, chlorides, or oxychlorides are heated in air. The V_2O_5 dissociates into VO_2 and oxygen at temperatures only slightly above its melting point (690°C). Numerous vanadates can be formed. Also, a variety of vanadium sulfates have been prepared.

Table 3.2. BOILING POINTS OF VANADIUM HALIDES AND OXYHALIDES[1]

Compound	Formula	Boiling point, °C
Vanadium tetrachloride	VCl_4	148.5^{755mm}
Vanadium oxychloride	$VOC1$	127
Vanadium oxytrichloride	$VOC1_3$	126.7
Vanadium trifluoride	VF_3	Sublimes
Vanadium oxytrifluoride	VOF_3	480
Vanadium pentafluoride	VF_5	111.2^{758mm}

3.1 REFERENCE FOR SECTION 3

1. **CRC Handbook of Chemistry and Physics, 51st Ed. Weast, R. C. (ed.). Cleveland, Chemical Rubber Company. 1970.**

4. SAMPLING, PREPARATION, AND ANALYSIS

4.1 INTRODUCTION

This section reviews the various analytical techniques that have been used to measure vanadium. It is intended to be representative of recent trends in such analytical methodology. Some general comments will also be given on quantitative aspects of procedures, which are related to accuracy. Athanassiadis[1] has collected relevant information on analytical methods for vanadium that will not be repeated here.

The importance of accuracy in the measurement of pollution is apparent when one attempts to compare analytical results among techniques and laboratories. Accuracy is critical if analyses are to be interchangeable among laboratories. Interchangeability of results is essential in work concerned with establishing threshold limits of pollutants, defining background concentrations, and defining degrees of toxicity.

Few analytical procedures applied to trace metals in pollution materials have been subjected to thorough or even adequate error analysis to permit rigorous definition of limits of accuracy. Although the American Public Health Association publication *Methods of Air Sampling and Analysis*[2] has a section entitled "Precision and Accuracy", for every method described, without exception, accuracy is not delineated — precision is covered; accuracy is not.

Rigorously defined, accuracy requires not only that all sources of significant systematic error be identified and quantitated, but also that the analytical system be in statistical control, as defined by Natrella.[3] Reasonable quantitative validation after the use of well-established procedures can result in useful comparison of analytical results among techniques and also among laboratories.

The more common methods for quantitative validation available to the analyst are listed below in approximate order of preference (within one or two rank positions); that is, Method 1 is least subject to inaccuracies, and Method 7 is most subject to inaccuracies.

1. Use of standard reference materials certified by a recognized standardizing agency or by industrial suppliers of specific materials.
2. Cooperative analyses involving several laboratories and several techniques (round robin).

3. Absolute analyses based on theoretical mathematical relations.
4. Method of standard additions using solutions.
5. Synthesized standards using solutions.
6. Same as Method 4, but using blended powders.
7. Same as Method 5, but using blended powders.

In addition to these seven, a family of radiochemical techniques[4] is in use — for example, the isotope dilution method. Although these methods are not truly quantitation procedures, they are important in this context because they provide highly useful means for minimizing some analytical inaccuracies.

Lack of certified standard samples for trace metals in pollution materials precludes the use of Method 1, standard reference materials, in many cases. The National Bureau of Standards either has issued or is planning to prepare some biological materials certified for trace metal content, but vanadium is not included. Furthermore, the availability and long-term preservation of standards applicable to the broad range of pollution samples will be very limited in the near future. Another problem in calibration lies in obtaining a true blank wherein the component to be measured is absent.

Method 2, cooperative analyses, requires a great deal of time, effort, and expense. This approach provides an opportunity to establish error limits under more realistic conditions than is feasible in a single laboratory. This cooperative work is not conducted primarily to establish accuracy. The round robin approach is more often conducted to establish uniform operating practices in several laboratories and to minimize the bias between laboratories using specified analytical procedures. In most cooperative work, the accuracy of the method is presumed to be established before distribution of samples to the cooperating laboratories. Nevertheless, a well-conducted round robin can reveal sources of analytical bias that bear on accuracy.

Method 3, absolute theoretical analyses, is exemplified by analyses based on the proved applicability of such theoretical relations as Beer's Law in colorimetry or atomic absorption, the Nernst equation in electrochemical procedures, and the Ilkovic equation in polarography. Analytical procedures that follow such relations are generally more amenable to good quantitation than completely empirical methods, provided that interferences are carefully defined.

The method of standard additions, Method 4, is one of the more powerful techniques for minimizing systematic errors in analysis. Shatkay[5,6] has presented mathematical analyses of the method of standard additions and of a similar technique, the method of successive dilutions, including a discussion of the assumptions and limitations of these methods that are often overlooked in their application.

Methods 4 through 7 involve synthesis of standards by blending either solutions or powders. Standards made from solutions are preferred over mixtures of solids. The achievable accuracy of this procedure depends on the close simulation of the standards to the samples. The more accurately the composition of the sample is known, the better its simulated composition can be. Synthesized solid standards are widely used, especially in emission spectroscopy and spark-source mass spectroscopy. This technique is subject to uncertainties that are exceedingly difficult to resolve. A major problem with solid materials not previously treated by dissolution is that the physical forms of the standard materials are not always identical with the form of the specific chemical element sought in the unknown sample. Apparently, much greater discrepancies can occur because of chemical differences between standards and samples. Diluting the sample in a uniform matrix can reduce some of the matrix (accompanying materials) effects. In spite of the highly utilitarian nature of these dilution methods, it is questionable whether they can be classified as quantitative without considerable supporting evidence as to their accuracy and their sensitivity to variations in sample matrix.

Any cursory survey of published analytical techniques will reveal that most offer only minimal evidence for inferring accuracy. Because this problem will undoubtedly persist, it is especially important for analysts and researchers concerned with the biological effects of atmospheric pollutants to be aware of the problems of proving accuracy and thus avoid some of the pitfalls in regard to quantitation. This is especially true in the interim before standardized procedures can be validated.

Inadequate control of blanks might be the most common cause of systematic errors when dealing with nanogram concentrations. Robertson[7] and other investigators have surveyed trace metal concentrations in glass and plastic containment materials, organic and inorganic reagents, wiping tissues, and other materials. Vanadium has not been detected in any of the commonly used reagents or plastic containment materials by the most sensitive detection techniques. Thus, at present levels of detectability, vanadium appears to be one of the least troublesome elements with respect to contamination. However, vanadium is also among the least concentrated elements in pollution materials. In the interest of accuracy, therefore, careful control must be exercised to avoid contamination from unsuspected sources.

4.2 ENVIRONMENTAL SAMPLES

4.2.1. Air

4.2.1.1. Sampling Particulate Matter—Among the major sampling techniques available for atmospheric particulates are absorption by liquid impinger, electrostatic precipitation, and filtration. The absorption method has pronounced drawbacks. Absorption may be far from complete, and many particulates, especially those of smaller diameters, may not be wetted and may pass out unabsorbed. The greatest disadvantages of electrostatic precipitation are the need for electric power, which may not always be available, the potential for formation of noxious gases, and the complexity and cost of the equipment. In more recent years, filtration methods have been used because of ease of operation and the availability of a variety of pure filtering materials. The filtering materials must be chosen with consideration for trace metal analysis of air samples, since elemental impurities may influence analytical results and complicate sample preparation. A typical glass-fiber filter, such as the type used routinely in the high-volume samplers, contains about 0.03 $\mu g/cm^2$ of vanadium as determined by atomic absorption spectroscopy[8] and 0.025 $\mu g/cm^2$ as determined by emission spectroscopy.[9] Because the blank value for any element may vary considerably with the batch, the filter blanks should be determined regularly. For sampling by NASN, flash-fired, glass-fiber filters were selected for low and uniform background.[10] Ashless paper filters and organic membrane filters that contain very small amounts of vanadium are available. Nevertheless, the size and nature of glass-fiber filters have made it possible to use a high-volume sampler and sample at an airflow rate of 1.5 to 1.6 m^3 per minute. When operating for 24 hours at this sampling rate, an adequate sample will be obtained even in an atmosphere that has little suspended particulate matter.

Although the method of sampling is important, it is even more important that the sample collected be representative of the original atmosphere. Since some of the vanadium halides have low boiling points, it is likely that there is some vanadium in the atmosphere in vapor form. Any sampling, therefore, should include atmospheric vapors.

4.2.1.2. Sample Preparation—Sample preparation affects both economy and accuracy of analyses. The ideal approach is direct analysis with no pretreatment at all, as carried out in some of the analytical procedures to be discussed. However, direct analysis is not always possible and might even be undesirable because of the difficulty in compensating for matrix effects through synthesis of standards.

Most analyses of particulate matter collected on paper filters involve some sort of sample preparation, usually mineralization through ashing of the sample. This can be accomplished in a muffle furnace[11-14] at temperatures between 400° and 650°C; by digesting in hot acid mixtures, such as nitric and perchloric acids;[14-16] or by ashing in an electrically excited oxygen atmosphere with a low-temperature asher (LTA).[17-20] The primary concerns in the ashing operations are the loss of metals by volatilization, metal contamination, and convenience of the procedure.

In recent years, the LTA method has gained preeminence over other ashing procedures for organic materials of all types.[18-22] It is superior from the standpoints of minimal concentration and ease of operation. Data reported for elements whose volatilities are comparable with those of vanadium and vanadium compounds suggest that no significant loss of vanadium occurs under normal conditions using the LTA.

4.2.1.3 Analysis by Atomic Absorption—The atomic absorption technique, although basically a single-element method, is advantageous because it is available, simple, and inexpensive. Vanadium forms thermally stable oxides that are only partially dissociated in the flame. Therefore, the hottest flame in common use, the nitrous oxide-acetylene flame, is used for routine measurements. The detection limit is 0.01 $\mu g/m^3$ for vanadium using the absorption mode, assuming a 2000-m^3 air sample.[20] Using the more sensitive emission mode, a detection limit of 0.005 $\mu g/m^3$ would be expected.[23] Although the lower detection limit is achieved in the emission mode, the absorption mode has been applied more often in routine analysis.

The elements that cause interferences are aluminum and iron.[24] Aluminum can be controlled by adding an excess of aluminum (200 $\mu g/ml$) to the samples and standards. The iron interference is negligible in concentrations found in ambient air. The estimated accuracy of the method is ±4.5 percent.[24]

In an application of atomic absorption analysis to airborne particulates, Kneip et al.[19] reported vanadium concentrations of 0.115 $\mu g/m^3$ in a nonurban area of New York, and 1.46 $\mu g/m^3$ in the Bronx area, New York City. The detection limit for vanadium was reported as 0.094 $\mu g/m^3$ for a total air volume of 5000 m^3, which is equivalent to a relative detection limit of about 10 $\mu g/ml$. In comparison, Morgan and Homan[13] reported a detection limit for vanadium in airborne particles to be 0.0018 $\mu g/m^3$ for a total air volume of 500 m^3 in 8.9 ml of solvent, or about 0.1 $\mu g/ml$. Atomic absorption spectroscopy[25] has been used to measure trace levels of vanadium in the atmosphere of eastern Massachusetts.

Flameless atomic absorption techniques that have received increasing attention are the tantalum ribbon atomizer;[8,26-28] heated graphite atomizer,[29] carbon rod atomizer,[30] and graphite furnace technique.[31,32] Advantages of these techniques are excellent sensitivity at nanogram and picogram levels and the need for an extremely small amount of sample solution — 10 microliters or less for a single measurement. Improvement of sensitivity over conventional flame mode by two or three orders of magnitude is not unusual.

Quickert et al.[32] and Hwang[8] indicate that the use of the conventional flame mode of atomic absorption spectroscopy is not sufficiently sensitive to measure many of the elements in atmospheric particulate matter, given a collection volume of 2000 m^3 through a high-volume air sampler and final elemental concentrations of 0.05 $\mu g/ml$ or less in solution. In measuring vanadium in airborne particulate matter using the flameless mode, Quickert et al.[32] found the sensitivity to be 0.0004 $\mu g/m^3$; with the flame mode, the same test sample showed a sensitivity of 0.025 $\mu g/m^3$.

Atomic absorption analysis for metals is limited to those elements for which hollow cathode lamps are available. The inability to measure more than one element at a time and the requirement that the sample be in solution are more severe limitations. Because air pollution is the result of high-temperature combustion, some of the particulate matter may be quite refractory and difficult to dissolve for atomic absorption analysis.

4.2.1.4 Analysis by Colorimetry — A variety of colorimetric methods are available for detection of vanadium. They are generally applicable to pollution materials with the use of suitable masking agents and extraction procedures to separate vanadium from interfering species. A number of colorimetric reagents, not necessarily applied to pollution materials, are listed in Table 4.1.[33-48]

A very simple, inexpensive, and specific method for determination of vanadium in air is the ring-oven technique; it uses the reagents salicylideneanthranilic acid[49] or 8-hydroxyquinoline[48] with estimation by eye.

4.2.1.5. Analysis by Electrometric Methods — Methods based on electrolytic phenomena are highly diverse in application and include at least 13 distinct techniques. These methods have found very limited use in the determination of vanadium in pollution materials.

Currently, the area of most active investigation is that of anode stripping voltametry (ASV), which provides the advantages of preconcentration, reasonably good specificity, sensitivity, and simplicity. However, vanadium is one of the more difficult elements to determine by ASV because of the lack of a suitable reversible reaction. Therefore, detection of vanadium in the materials of interest by this technique has not yet been reported.

A polarographic method has been described for determination of vanadium in the air of the working environment in the alloy industry.[54] The method is sensitive to 1.5 μg of vanadium pentoxide per ml.

Vanadium has also been detected by other electrometric techniques, including potentiometry,[55] amperometry,[56,57] and coulometry.[58,59]

4.2.1.6. Analysis by Electron Optics — Tools for analysis by electron optics include the electron microscope, the electron microprobe, and X-ray diffraction. These tools, supplemented by light

Table 4.1. COLORIMETRIC REAGENTS FOR DETECTING VANADIUM

Reagent	Reference	Remarks
5-Amino-3-(3-chloro-2-hydroxy-5-nitrophenylazo)-4-hydroxynaphthalene-2,7-disulfonic acid (gallion)	33	$\epsilon \cong 8{,}700$
5-Amino-4-hydroxy-3-(2-hydroxy-3,5-dinitrophenylazo)naphthalene-2,7-disulfonic acid (picraminazo N)	33	$\epsilon = 12{,}400$
Unsaturated N-arylhydroxyamic acids[a] (23 complexes studied)	34	Nine complexes had $\epsilon \cong 1{,}500$
Cyclo-tris-7-(1-azo-8-hydroxy)-naphthalene-3,6-disulfonic acid (calichrome)	37	—
Diaminobenzidine	38	—
8-Hydroxyquinoline	39–43	—
Naphthalene-2,3-diol (2,3-dihydroxynaphthalene)	44	—
m-Nitro-N-phenylbenzoylhydroxyamic acid	45	Beer's Law followed from 0.2—11 μg/g
N-Phenylbenzoylhydroxyamic acid	34	$\epsilon = 4{,}650$
Phenylhydrazine-p-sulfonic acid[b]	46	—
N-Phenyl-3-styrylacrylohydroxyamic acid	34	Beer's Law followed from 0.7 to 8.4 μg/g; sens. = 0.0068 $\mu g/cm^2$
2,6-Pyridinedicarboxylic acid	47	Beer's Law followed up to 5 mg V/50 ml of solution
4-(2-Pyridylazo)resorcinol	48	—
Salicylideneanthranilic acid	49	Can be determined between 0.1 and 3 μg
N-o-Tolylbenzoylhydroxyamic acid	50	—
Tungstate	51–53	Sensitivity, 5 μg V_2O_5/5 ml

[a] Salicylhydroxyamic acid.[35,36]

[b] Vanadium catalyzes the oxidation of phenylhydrazine-p-sulfonic acid by sodium chlorate. A diazonium salt is produced that couples with 1-naphthylamine to form an azo dye.

The 8-hydroxyquinoline procedure[39] was adapted to air-filtered materials and specified as a tentative method by the Intersociety Committee on Methods of Air Sampling and Analysis. The 8-hydroxyquinoline reagent was also applied after extraction of vanadium with -benzoin oxime in chloroform.[42] This reagent has been used to measure levels of vanadium pollution in the air of Helsinki, Finland,[40] and at various Japanese manufacturing sites.[41]

The salicylhydroxyamic acid reagent has been used in measurement of air in Germany.[35,36]

microscopy, allow characterization beyond elemental analysis and into the area of morphologic, compound, and crystallographic identification, as described by Rhodes[60] and Blosser.[61] Although no vanadium compounds have yet been identified with these techniques, vanadium distributions within particulate matter samples from the Washington, D. C., area have been reported.[61]

4.2.1.7. Analysis by Emission Spectroscopy — Emission spectrochemical methods are differentiated on the basis of method of sample preparation, method of quantitation, and method of exciting the atomic spectra. As with instrumental methods of analysis in general, a wide variety of experimental procedures have been developed that represent trade-offs between simplicity and economy on the one hand, and precision and accuracy on the other. One of the simplest methods of analyzing filtered air samples by emission spectroscopy requires no pretreatment of the sample at all.[62] Although the repeatability of the method is adequate for monitoring concentration trends of some elements, its accuracy is unknown because calibration standards consisted of dried solutions of the various elements on filter paper. Quantitation of the method using air-filtered specimens analyzed by other means could also be used. Vanadium was not reported by this method.

A procedure has been reported for detecting vanadium and nine other elements in suspended particulate matter.[63] In this method, the chemical forms of the particles were destroyed by ashing and then fusing the ash with lithium tetraborate. The ground fusion material was then pelleted with graphite and subjected to a spark discharge. This technique tended to minimize inaccuracies caused by differences in chemical form between unknown samples and standards, assuming that the composition of the major elements in the standards was approximately the same as that in the samples. The mean vanadium concentration of 270 samples taken in metropolitan New York air was 0.17 $\mu g/m^3$.

Possibly the most often used emission spectrometric procedure for detection of metals in airborne particulate matter and in biological materials is a variation of the so-called universal method of analysis.[64] Many commercial laboratories use this general approach because it is economical. There are many variations of this approach, which basically involves diluting the ashed sample in a relatively pure powdered material in a sample: diluent ratio of about 1:10. Typical diluents are graphite, lithium carbonate, and lithium fluoride. The dilution reduces all samples to a relatively common matrix and therefore reduces some systematic errors caused by variation in sample composition. However, a fundamental uncertainty remains concerning physical forms of the various chemical species. Because it is not possible to place limits on this source of error, these methods require quantitative validation by other means.

The emitted light from a sample that has undergone the prescribed processing can be received either by a photographic plate[65-69] or film, or by a photomultiplier tube.[10,70] In the first method, the plate is developed, and the spectral lines are investigated. The darkness of the lines is measured, and the concentration of the element being analyzed is calculated. When measured by this technique, vanadium concentrations in Osaka, Japan,[65] ranged from 0.05 to 0.55 $\mu g/m^3$ In another study of 11 Japanese cities,[67] the average concentrations ranged from 0.014 to 0.269 $\mu g/m^3$.[67] Tabor and Warren[68] measured 17 metals in 20 U. S. communities by this technique. They reported that a large percentage of samples contained less than the minimum detectable amount of the metals, and they pointed out the need for a more sensitive method. Tani[67] reported that the photographic technique is time-consuming and not desirable for routine measurements. The data obtained in recent years by NASN[10,70] have been acquired through the use of a direct-reading emission spectrometer. For 1970 NASN samples analyzed in 1972, duplicate measurements of vanadium in 32 cities gave an average concentration of 0.096 $\mu g/m^3$, with a relative standard deviation of 9.3 percent.[70] Matrix effects from the widely varying samples were reduced by use of a 2 percent lithium chloride buffer and an indium internal standard. The detection limit reported for vanadium[20] is 0.01 $\mu g/m^3$ by atomic absorption spectroscopy and 0.003 $\mu g/m^3$ by emission spectrography.

Disadvantages in the use of emission spectroscopy for metals analysis include the complicated methodology required to obtain reliable data and the detection limit, which prohibits analysis for low-level concentrations of some metals.[20] In addition, the instrument is expensive to buy and requires highly trained technical personnel to operate.[20] However, when samples are to be analyzed routinely for more than six metals, the emission spectrograph saves time and effort.

4.2.1.8. Analysis by Fluorimetry — For determination of vanadium in solution, a simple method has been reported that measures the fluorescence intensity produced on reaction with benzoic acid in the presence of zinc amalgam.[71] The fluorescence intensity was a linear function of concentration for 0.0005 to 0.4 $\mu g/ml$ of vanadium· Most common ions, except Fe^{3+} and Ti^{4+}, did not interfere. Further studies are needed to devise better procedures for removal of the interferences. No practical application to vanadium measurement was reported.

4.2.1.9 Analysis by Gas Chromatography — Hyperpressure gas-phase chromatographic separations of organic vanadium compounds and their later detection are emerging from the developmental stage.[72,73] The method has found practical application in detection of trace metals in biological materials,[74] but no practical application has been reported for vanadium.

4.2.1.10. Analysis by Neutron Activation — Basically, neutron-activation analysis involves exposing a sample to a source of neutrons and detecting nuclear radiation emitted by the isotope of the element made radioactive by absorbing a neutron. For airborne particulate analysis, it is essential that samples be collected on filters low in trace elements. Filters found suitable for sampling include Whatman,[30] cellulose fiber paper,[75] millipore filters,[76] polystyrene filters,[77] membrane filters,[78] and ashless filter paper.

Radiation detection and measurement are by gamma-ray spectrometry, using either a thallium-activated sodium iodide detector, NaI(Tl), or a lithium-drifted germanium detector, Ge(Li). In samples that emit few gamma rays of different energies, the NaI(T1) detector is sufficient for quantitative determinations. This detector is relatively inexpensive ($5,000 to $6,000) and can be operated at room temperature with 1.5 to 2 times the counting efficiency of the Ge(Li) detector.[79] Resolution, however, is poor. In comparison, the Ge(Li) detector has excellent resolution, and there are few effects of other nuclides present. However, its counting efficiency is low, it is costly (more than $25,000),[79] and it requires constant cooling.

Neutron activation can be considered either a destructive or nondestructive technique, depending on whether the sample can be counted after irradiation and cooling or whether chemical separation of isotopes is required (because of overlapping peaks) before the sample is counted. The main objective is isolation of the peaks. In some cases, separation is attempted before irradiation, but for purposes of trace-element analysis in air pollution, the possibility of element losses prohibits using such a measure.

In neutron activation of vanadium, the radioactive species is vanadium-52, with a half-life of 3.77 minutes and a gamma energy of 1.434 million electron volts[80] — a so-called short-lived isotope. The main interference in the analysis of vanadium is aluminum-28, which has a half-life of 2.27

minutes and is found in a ratio of 50 or more to 1 of vanadium in airborne particulates.[76] Bando and Imahashi[76] estimated the vanadium in dustfall deposits in two Japanese cities using a NaI(T1) detector; they overcame the aluminum interference by extracting, by means of chloroform, the vanadium from the irradiated sample as a 2-methyl-oxime complex. The extract was subjected to gamma ray spectrometry, the absorbance was measured, and the vanadium was estimated by seeking the chemical yield by photometry. The time required for analysis was 10 minutes, and the average chemical yield was 95 percent. Concentrations of vanadium found in the two cities using this method ranged from 0.13 to 0.98 $\mu g/m^3$.

Following irradiation of samples from northwest Indiana and counting with a Ge(Li) detector after a 3-minute decay period, Dams et al.[77] found the minimum concentration for vanadium in urban air to be 0.002 $\mu g/m^3$ as compared to 0.0032 $\mu g/m^3$ using emission spectrography. The sensitivities obtained for nonurban samples were better than those obtained for urban samples with either method. Rancitelli et al.[81] studied air samples from 14 cities by neutron-activation analysis using a 10-minute decay period to remove the major interference (aluminum-28) before counting on a Ge(Li) spectrometer, and compared the data with samples analyzed by X-ray fluorescence. The authors report their accuracy and precision for a single determination to be better than 10 percent for most elements in air filters. Using the Ge(Li) detector, Martens et al.[75] found vanadium concentrations in San Francisco to range from 0.0027 to 0.012 $\mu g/m^3$. The effectiveness of this method has also been demonstrated in studies of the Livermore Valley, California, and of Niles, Michigan.[82,83]

Neutron-activation analysis is most important in air pollution studies because of its high sensitivity for many elements and the fact that 20 or more elements can be observed in a single sample. However, this technique is inadequate for determining lead or phosphorus.[84] In addition, to realize the full capability of measuring 33 elements, total cooling periods of up to 30 days are required.[77] This introduces a delay between receiving the sample and obtaining a complete elemental analysis.

4.2.1.11. Analysis by Spark-Source Mass Spectrometry — The spark-source mass spectrometer has been used for multielement analysis of both airborne particles and biological samples.[12,14,17] The sample preparation in all cases consisted of ashing the sample and pelletizing the ash with graphite to achieve the necessary electric conductivity.

A more recent innovation is the use of electric detection with the spark-source mass spectrometer. This new method promises to simplify the technique and to improve the detection repeatability.[85]

By the mass-spectrometric technique, the vanadium concentration in New York City air was determined[11] to be 1.9 $\mu g/m^3$. The particulate sample was collected on nitrocellulose filters that were ashed at 450°C and pelleted with graphite. Relative sensitivity factors and metal losses in the ashing step were not taken into account.

4.2.1.12. Analysis by X-Ray Fluorescence — X-Ray fluorescence is an attractive method for analysis of air pollution particulate samples for a number of reasons. The technique is nondestructive and the sample is thus retained for further analysis. Detectability is fairly uniform across the periodic table; all elements from atomic number 11 upward can be analyzed, and 10 or 20 elements can be analyzed simultaneously with commercially available equipment.

Disadvantages of the X-ray fluorescence technique include the high initial cost — ($60,000 to $200,000) — of a fully automatic, computerized instrument. Cost can be cut in half if samples and data are handled manually. There are problems in preparing calibration standards representative of actual samples. Interelement effects may lead to complications.[8] Errors as large as a factor of 2 can be introduced into the X-ray analytical result if a significant number of the particles collected are larger than a few micrometers.[86] At this time, the only solution to this problem is use of size-fractionated samples.

The X-ray measurements for air pollution samples reported in the literature were obtained from the use of either energy dispersion or wave length dispersion instruments. Several types of excitation sources have been evaluated:

- Lithum-drifted silicon [Si(Li)] detector with radioisotopes[81,87] or low-powered X-ray tube[88] and fluorescer excitation for energy dispersion.[89,90]
- Radioisotope-fluorescer assembly with a solid-state detector.[91]
- Radioisotopes and a solid-state detector.[92]
- Proton[93] or alpha particle[94] excitation.
- Crystal spectrometers with X-ray tube excitation for wavelength dispersion[9,95-97]

Cooper[98] has compared various methods of exciting samples for subsequent X-ray analysis.

Filter materials used to collect particulate samples for analysis by X-ray must be as pure as possible and, with the particulate, constitute a sample that is easily penetrated by X-rays so that no matrix effect will result. Certain types of millipore filters and Whatman filter paper have been found to satisfy these requirements.

Birks et al.[9] used a crystal diffraction spectrometer and an analysis time of 100 seconds per element in the determination of 12 elements from stationary source sites. The detection limit for vanadium on Whatman 41 filters was reported as 0.029 $\mu g/cm^2$, which corresponds to a detection limit of 0.003 $\mu g/m^3$ for a 24-hour sampling period. Extensive measurements of suspended particulate matter were made at 38 stations in Texas by Rhodes et al.[87] through the use of energy-dispersion instrumentation consisting of three radioisotope sources for excitation and a Si(Li) detector to receive the signal. Samples collected on Whatman 41 filters were analyzed for 900 seconds, and concentrations for 15 elements were given. Low values (less than 0.008 $\mu g/m^3$) were reported for vanadium. For particulate samples collected on 0.8 μm millipore filters and analyzed for 900 seconds using an energy-dispersive system consisting of an X-ray tube[90] with three separate secondary fluorescers, Goulding and Jaklevic give the detection limit for vanadium as 0.006 $\mu g/m^3$. The authors mention that the sensitivity would be improved if the system were optimized for each element by filtering the X-rays.

Future experimental studies of X-ray fluorescence are needed to overcome the inadequacies of this technique and extend the application.

4.2.2. Water

Many laboratories use automated sampling techniques to determine metals in water by direct aspiration. For trace-metals analysis, however, the direct aspiration method does not generally have the required sensitivity; concentration of the metals is required.

One such method is based on complexing the metal with ammonium pyrrolidine dithiocarbamate and extracting the complex with metyl isobutyl ketone (MIBK). The MIBK phase is then separated and aspirated into the flame of an atomic absorption spectrophotometer.[99] Although Goulden et al.[99] report no values for vanadium using this technique, further studies could show it to be valuable. Microgram quantities of vanadium in lake water (5 μg/liter) have been detected by atomic absorption after extraction with 5,7-dichloro-8-hydroxyquinoline.[100] The flameless atomic absorption method has also been used for trace metals in water.[28]

A method with a sensitivity of 0.1 to 8 μg/liter has been developed for analysis of vanadium in water by measuring the catalytic effect it exerts on the rate of oxidation of gallic acid by persulfate in acid solution.[101] The extent of oxidation of gallic acid is proportional to the existing concentration of vanadium. Vanadium is determined by measuring the absorbance of the sample at 415 nanometers and comparing it with standard solutions. Halide interference is reduced by dilution or complexation with mercuric ion. The minimum detectable concentration is 0.025 μg.

Neutron-activation analysis[102] has been applied to the determination of vanadium (1 to 10 μg/liter) in natural waters by collecting vanadium on an ion-exchange resin and later irradiating the nitric acid eluate.

The previous section concerning methods for analysis of air for vanadium suggests that techniques such as colorimetry, fluorimetry, and emission spectroscopy should provide further means of measuring vanadium in water.

4.2.3. Soil

4.2.3.1 Soil Sampling — The method of sampling used in determining the vanadium content of the soil, as with any other substance, is critical if an accurate appraisal is to be obtained. Random sampling noting the type of rock or the composition of the soil is of extreme importance because the vanadium content will vary according to the sample's makeup.[103,104]

4.2.3.2. Analysis Procedures — Spectrochemical analysis, atomic absorption, and neutron activation have been the chief methods used in analyzing the soil for its vanadium content. The sensitivity of the spectrographic method is 3 μg/g; however, different analysts using the same material have not always achieved the same results.[105] Kemp and Smales[105] have shown that through neutron activation, it is much easier to duplicate results (Table 4.2). These three methods of analysis have been discussed in detail earlier. Sample preparation for soil analysis, however, differs from the methods described for other media.

Spectrochemical analysis (emission spectroscopy) involves preparation of the samples by digestion and then chemical pretreatment. The mixture thus prepared is used for arcing.[106] During preparation of samples, extreme caution is necessary to prevent trace-element contamination. The procedure as outlined by Shimp et al.[106] is precise to ±10 percent. Preventing contamination of the element being analyzed is of utmost importance if analysis is to be accurate.

Table 4.2. ANALYTICAL RESULTS FOR "STANDARD" ROCKS[105]
($\mu g/g$)

"Standard" rock	Range of other workers	"Recommended" value	90% confidence interval	This work	
				Individual determinations	Average
G-1	8–38	18	21 ± 7	13.4, 13.2, 12.9, 13.1, 13.0, 13.0, 13.0, 12.7	13
W-1	120–340	240	255 ± 55	250, 244, 244, 244, 247, 244	246

Atomic absorption analysis requires ashing and dissolving of samples. The flameless furnace-vaporization technique is best for solid samples.[107] The atomic absorption method detailed by Goeke[108] for detecting vanadium in ores has a sensitivity of 0.2 ppm for 1 percent absorption.

Neutron activation has been used to determine the content of vanadium in rocks and meteorites.[106] As indicated previously, Kemp and Smales[105] have shown that, through use of neutron activation, it is possible to come closer to duplicating results. They indicate that molybdenum and technetium are the only interfering elements, and then only when the vanadium content of the sample is low. The sensitivity of the methods for rocks and meteorites is not indicated; however, a vanadium content of less than 0.2 $\mu g/g$ average is listed in a table showing the vanadium content of meteorites.

4.3 BIOLOGICAL MATERIALS

4.3.1. Sampling

Studies are underway for collecting biopsy and autopsy human tissue specimens for the purpose of systematically determining baseline pollutant levels in different organs as a function of age, race, smoking status, residence, and occupation.[109,110] This program is an effort to determine the temporal and geographic patterns for selected populations to enable assessment of pollutant body burdens and to attempt to correlate these burdens with statistically observed disease patterns. These epidemiologic studies are based on a minimum of 1000 individuals in each area. Short- and long-term health indicators are employed, such as frequency and aggravation of asthma attacks and other evidence of illness or mortality that can be linked to pollutant exposures.[110,111]

Tissue sampling includes several steps: types of tissues (part of anatomy), size and amount, containment, transport, preparation for analysis, and short- or long-term storage. Each step is part of the overall analytical process and may affect the integrity of the trace elements in the sample.[112] Written sampling protocols are available to ensure uniformity in collection by various field sampling stations.[110,113,114]

Easily collected tissue specimens such as blood, urine, and feces primarily reflect short-term exposure changes and are incomplete indicators of total residue of vanadium in the body. These types of samples are valuable in studying biological residence times of vanadium in the body.

In humans, vanadium accumulates in the lung. It also has an affinity for fatty tissues, bone, and keratinous tissues such as hair and skin. The usefulness of hair as an indicator of both short-term acute exposure and long-term chronic exposures to vanadium and other trace metals has been or is being studied by several investigators.[109,111,115,116] Hair has been established as a useful indicator of acute exposures to mercury, lead, cadmium, and vanadium. At vanadium levels normally found in urban and industrial areas, hair is of marginal value as an indicator of exposure or body burden of vanadium.

Under the EPA CHESS (Community Health and Environmental Surveillance System) program, arrangements are made with pathology departments of local hospitals for collecting autopsy and biopsy tissues. Sampling protocols, which have been prepared for pathologists, include procedures for collecting 19 different tissues. Surgical tools to be used are specified to avoid contamination during excising. There is also a questionnaire to be completed for each set of autopsy tissues to provide supporting information such as medical, occupational, and smoking history; unusual dietary habits; medicinal intake; cause of death; and other information useful in assessing possible sources of intake of a trace metal. Specific instructions are provided for container design, labeling, shipping, and storage for tissue specimens.

Before a tissue specimen is contained, preserved, and stored, a decision must be made on whether to homogenize, lyophilize, or otherwise preserve a whole organ or to select a particular area or part of an organ or tissue. The latter would enable a more precise assessment of the site of residence of vanadium or other elements. If the tissue specimen is homogenized, it should be uniform. This is important if the sample is analyzed by several laboratories. Inconsistent findings will result if fragments of tissue, bone, etc. are dispersed or settle from a homogenized sample.

4.3.2. Analysis

The procedure used to prepare a tissue specimen for analysis depends on type of sample, element to be determined, and analytical procedure or instrument to be used. Problems of analysis are similar to those of other biological tissues (foods, plants, etc.) where other elements can interfere. The multielement composition of biological tissues provides the analyst with technical problems.

Methods and instruments that are of major interest for analyzing tissues for vanadium include:
- Neutron-activation analysis — radiochemical and instrumental.
- Atomic absorption spectrophotometry — flame, graphite furnace, and flameless.
- Emission spectroscopy — dry ashing.
- Optical colorimetry.

These methods are applicable to the analysis of other biological materials.

Neutron activation is a rapid and sensitive technique for detection of vanadium in biological materials. The rapid decay (half-life 3.8 minutes) of the induced radiovanadium, V-52, is a problem.

Preparation of a hard tissue is particularly difficult in the short time available for counting radiovanadium.

Neutron-activation analysis of vanadium allows estimation on the order of 10^{-9} gram. This sensitive technique is reduced by elapsed time between irradiating specimens in the reactor and counting. Investigators have irradiated biological specimens prepared by acid digestion in a reactor and made counts in 8 to 10 minutes (two half-lives) of radiovanadium, V-52. This permitted measurement of vanadium in biological materials within the range of ±5 percent.

Söremark[117] similarly used neutron-activation analysis to determine vanadium content of biological specimens. Generally, he reported lower values for vanadium than did Schroeder[116] using emission spectroscopy. Söremark also employed autoradiography to determine distribution patterns in fish. Autoradiograms of whole-body sections were taken after fish were kept for 3 days in water containing vanadium-48.

Hair and placental specimens collected under the CHESS program are analyzed by emission spectroscopy.[115] Atomic absorption scpectroscopy has been used to measure trace levels of vanadium in a few urban woody plant species in New Hampshire and Vermont.[118]

4.4 STATIONARY SOURCE SAMPLES

4.4.1. Sampling

No direct-reading instruments for field and continuous or semicontinuous application are available for measuring vanadium in stack emissions. Samples of dust or fumes are collected with filter paper samplers, electrostatic precipitators, and/or impingers, and then analyzed in the laboratory.

4.4.2. Analysis

Analytical techniques relying on instrumental methods have been applied to measure vanadium in various media, including particulate matter collected from ambient air. The predominantly used techniques are colorimetry, atomic absorption spectrophotometry, atomic fluorescence, emission spectroscopy, X-ray fluorescence, neutron-activation analysis, and mass spectrometry. Most of these methods are based on analyses only of particulate collected on filters (paper, organic membrane, or glass fiber), as they suffer from potential sample loss by vaporizing volatile compounds in stack emissions when large volumes of air are drawn over the collected particulate matter. This effect would be noticed readily when collecting compounds that are obviously volatile. The effect also could occur in special cases — for example, the volatilization of chromic oxide can be enhanced by forming chromium trioxide in an oxygen atmosphere, particularly in a dynamic system with a considerable flow of gas. Some of this loss can be minimized by a tandem arrangement of filter and impinger containing a collecting liquid. The vaporization losses on filters could be moderately high when sampling heated stack gases.

The following sections discuss technology related to analytical methods that can be applied to the continuous or short-term measurement of metals from stack emissions. Although a variety of techniques are presented, the most attractive methods for general application (vapor and particulate), based on selectivity and sensitivity, are atomic absorption and emission spectroscopy. Others, such as X-ray fluorescence, may find application in measuring only particulate matter.

4.4.2.1. Atomic Absorption Spectrophotometry — Because of moderately high sensitivity and good specificity, atomic absorption spectrophotometry has been used to measure trace quantities of metallic elements collected from ambient air after dissolution of the particulate in acid. The major problem in applying the technique to continuous monitoring of stack emissions for vanadium is the inability to handle refractory compounds and elements that readily form stable compounds, such as vanadium oxides, in the flame. Serious matrix and interelement effects are observed.

Matrix effects and compound formation are most common in air- or oxygen-acetylene flames. As a consequence, direct aspiration of air-contaminated gas into the flame has not been used to any large degree for continuous monitoring of metallic elements in air or stack emissions. Such a system can be developed, but the estimated detection limit would only be about 1 mg/m^3 without a preconcentration step.

A modified atomic absorption spectrophotometer for simultaneous determination of four selected elements was described by Zwiebaum and Moorhead.[119] The system was designed for continuous monitoring of several elements concentrated from the atmosphere into a small-volume air stream. The air stream with enriched sample content is fed directly and continuously into the burner nebulizer. The highest sensitivity, minimal matrix, and minimal chemical interferences are attained with nitrous oxide as the oxidant and with a nitrogen sheath gas to restrict interaction of ambient air with the chemical species in the flame.

Interelement effects can also be eliminated by using a high-energy plasma to atomize the elements being measured. The major problem in applying this technique is to maintain high levels of ground-state atoms but low ionization efficiencies in the plasma. Energy from the plasma may ionize the element sought, thus reducing the population of ground-state atoms, which must be high to attain high sensitivities for the atomic absorption phenomenon.

4.4.2.2. Emission Spectroscopy — Although emission spectroscopic techniques have been used extensively to measure the metal content of particulates collected by NASN,[120,121] no data are available for continuous, flow-through emission spectroscopic studies on airborne or stack emissions of vanadium. Section 4.2.1. outlines the uses of emission spectroscopy for the analysis of filtered samples.

4.4.3. Evaluation

At this time, there is no continuous monitoring system for measuring vanadium from stationary source emissions. A continuous monitoring system for vanadium in stack gas is possible if the element is kept suspended in the gas and the gas is then introduced as part of the oxidant or fuel in the flame or high-energy discharge of the excitator source of the spectrometer. To date, most laboratory techniques have been applied to integrated samples of particulate matter collected on filter paper from ambient air.

High sensitivities and the attendant low detection limits of atomic absorption and emission spectroscopy make these methods most attractive for continuous or short-term intermittent monitors. With vanadium, the major problem in using the absorption or emission spectroscopic techniques is the interelement or compound formation effects.

With atomic absorption spectrophotometry, the interelement or compound formation effects are minimized or eliminated by using a nitrous oxide-acetylene flame. The best results are obtained with a sheath gas, which is an inert gas such as nitrogen, to exclude oxygen from the flame. Obviously, in ambient air or stack emissions, elimination of the oxygen is extremely difficult. One possible way to eliminate the oxygen is to pass the stack gas or air over hot carbon to produce carbon monoxide.

The use of an inert gas plasma as an atomization source for atomic absorption spectrophotometry will also minimize interelement effects if a reducing atmosphere is maintained by introducing hydrogen gas. An additional problem related to the use of a plasma to provide the atomization for atomic absorption measurements is the possibility of overexciting the element sought and causing its emission.

With a high-resolution semiconductor detector, X-ray emission techniques with conventional X-ray target tubes and with radioisotope sources can be used on an intermittent basis to determine vanadium in particulate collected on filters. Care must be taken to measure thin film deposits so as to minimize matrix effects. Also, the background of secondary emission of X-rays from the filter media can be a major interference.

Radioisotope sources for X-ray emission measurements provide a high degree of portability and simplicity of ancillary equipment. When applied to monitoring particulate collected on filters, good accuracy and repeatability of measurements are obtained. The major limitations in applying the X-ray techniques are the time necessary to collect sufficient particulate matter on the filter and the potential loss of volatile emissions containing the element sought.

Nondestructive neutron-activation analysis for vanadium can be performed on particulate matter. The major problem is interference from radioactive isotopes of other elements.
Fast neutrons from an isotopic source give much shorter irradiating and cooling times, compared to other neutron sources. There is some sacrifice of sensitivity, but the technique can detect vanadium in approximately 30 minutes.

Continuous, on-stream (aqueous solutions), neutron-activation analyses for vanadium have been performed with isotopic sources at the ⩽100 ppm level in 5- to 10- minute cycles.

Any technique relying on collection of particulate matter on filters from relatively large volumes of air or stack gases risks the loss of volatile compounds.

Data are needed on the particle size distribution of vanadium emissions to supply input into air modeling schemes designed to determine the fate of the emitted material.

4.5 REFERENCES FOR SECTION 4

1. Athanassiadis, Y. C. Preliminary Air Pollution Survey of Vanadium and Its Compounds; A Literature Review. U. S. Department of Health, Education, and Welfare, Public Health Service, National Air Pollution Control Administration, Raleigh, N. C. NAPCA Publication No. APTD 69-48, October 1969, 91 p.

2. Methods of Air Sampling and Analysis. American Public Health Association, Intersociety Committee, Washington, D. C., 1972.

3. Natrella, M. G. Experimental Statistics. National Bureau of Standards Handbook 91, National Bureau of Standards, Washington, D. C., 1966. Errata, 3 p.

4. Trace Analysis — Physical Methods, Morrison, G. H. (ed.), New York, Interscience Publishers, Inc. 1965. 582 p.

5. Shatkay, A. A Critical Analysis of the Method of Successive Dilutions in Photometry. Anal. Chim. Acta. *52*:547, 1970.

6. Shatkay, A. Photometric Determination of Substances in the Presence of Strongly Interfering Unknown Media. Anal. Chem. *40*:2097, 1968.

7. Robertson, D. E. Role of Contamination in Trace Element Analysis of Sea Water. Anal. Chem. *40*:1067, 1968.

8. Hwang, J. Y. Trace Metals in Atmospheric Particulate and Atomic Absorption Spectroscopy. Anal. Chem. *44*:20A, 1972.

9. Birks, L. S., J. V. Gilfrich, and P. G. Burkhalter. Development of X-ray Fluorescence Spectroscopy for Elemental Analysis of Particulate Matter in the Atmosphere and in Source Emissions. U. S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N. C. Publication No. EPA-R2-72-063, October 1972.

10. Air Quality Data for 1968 from the National Air Surveillance Networks and Contributing State and Local Networks. U. S. Environmental Protection Agency, Office of Air Programs, Research Triangle Park, N. C., Publication No. APTD-0978, August 1972. 243 p.

11. Brown, R., and P.G.T. Vossen. Spark Source Mass Spectrometric Survey Analysis of Air Pollution Particulates. Anal. Chem. *42*:1820, 1970.

12. Lambert, J. P. F., R. E. Simpson, H. E. Mohr, and L. L. Hopkins, Jr. Determination of Vanadium by Neutron Activation Analysis at Nanogram Levels to Formulate a Low-vanadium Diet. J. Assoc. Offic. Anal. Chem. *53*:1145, 1970.

13. Morgan, G. B., and R. E. Homan. The Determination of Atmospheric Metals by Atomic Absorption Spectrophotometry. U.S. Department of Health, Education, and Welfare, Public Health Service, National Air Pollution Control Administration, Cincinnati, Ohio. (Presented at Pittsburgh Conference on Analytical Chemistry and Applied Spectroscopy, February 1967.)

14. Yurachek, J. P., G. G. Clemena, and W. W. Harrison. Analyses of Human Hair by Spark Source Mass Spectrometry. Anal. Chem. *41*:1966, 1969.

15. Christian, G. D., and F. J. Feldman. Atomic Absorption Spectroscopy Applications in Agriculture, Biology and Medicine. New York, Wiley Interscience, 1970. 490. p.

16. Delves, H. T., G. Shepherd, and P. Vinter. Determination of Eleven Metals in Small Samples of Blood by Sequential Solvent Extraction and Atomic Absorption Spectrophotometry. Analyst. *96*:260, 1971.

17. Evans, C. A., Jr., and G. H. Morrison. Trace Element Survey Analysis of Biological Materials by Spark Source Mass Spectrometry. Anal. Chem. *40*:869, 1968.

18. Gleit, C. E., and W. D. Holland. Use of Electrically Excited Oxygen for the Low Temperature Decomposition of Organic Substances. Anal. Chem. *34*:1454, 1962.

19. Kneip, T. J., M. Eisenbud, C. D. Strehlow, and P. C. Freudenthal. Airborne Particulates in New York City. J. Air Pollut. Contr. Ass. *20*(3):144-149, 1970.

20. Thompson, R. J., G. B. Morgan, and L. J. Purdue. Analysis of Selected Elements in Atmospheric Particulate Matter by Atomic Absorption. Atomic Absorption Newsletter. *9*:53, 1970.

21. Welz, B., and E. Wiedeking. Bestimmung von Spurenilementen in Serum und Urin mit Flammenloser Atomisierung (Determination of Trace Elements in Serum and Urine Using Flameless Atomization). Fresenius' Z. Anal. Chem. (Wiesbaden). *252*:111, 1970.

22. Hambridge, M. K. Use of Static Argon Atmosphere in Emission Spectrochemical Determination of Chromium in Biological Materials. Anal. Chem. *43*:103, 1971.

23. Outline Guide for Analysis of Elements in Trace Amounts. Dow Chemical Company, Midland, Mich., Dow Interpretive Analytical Services, Vol. 2, No. 4, 1971.

24. Goeke, R. Determination of Vanadium in Ore Samples by Atomic Absorption Spectrophotometry. Talanta. *15*:871, 1968.

25. Barry, E. F., M. T. Rei, and H. H. Reynolds. Trace Nickel and Vanadium Determinations in the Atmosphere of Eastern Massachusetts by Atomic Absorption Spectroscopy. (Presented at American Chemical Society Fifth Northeast Regional Meeting, Rochester, N. Y., October 14-17, 1973).

26. Donega, H. M., and T. E. Burgess. Atomic Absorption by Flameless Atomization in a Controlled Atmosphere. Anal. Chem. *42*:1521, 1970.

27. Hwang, J. Y., P. A. Ullucci, and C. J. Mokeler. Maximization of Sensitivities in Tantalum Ribbon Flameless Atomic Absorption Spectrometry. Anal. Chem. *44*:2018, 1972.

28. Hwang, J. Y., P. A. Ullucci, and S. B. Smith, Jr. A Simple Flameless Atomizer. Amer. Lab. *3*:41-43, August 1971.

29. Fernandez, F. J., and D. C. Manning. Atomic-absorption Atomization With Use of a Heated Graphite Tube. Atomic Absorption Newsletter. *10*:65, 1971.

30. Amos, M. D., P. A. Bennett, K. G. Brodie, P. W. Y. Lung, and J. P. Matousek. Carbon Rod Atomizer in Atomic Absorption and Fluorescence Spectrometry and Its Clinical Application. Anal. Chem *43*:211, 1971.

31. Woodriff, R., and G. Ramelow. Atomic Absorption Spectroscopy with a High-Temperature Furnace. Spectrochim. Acta. *23B*:665, 1968.

32. Quickert, N., A. Zdrojewski, and L. Dubois. The Accurate Measurement of Vanadium in Airborne Particulates. Int. J. Environ. Anal. Chem. *3*:229-238, September 1972.

33. Zadumin, E. A., and A. I. Cherkesov. Photometric Determination of Vanadium with Picraminazo-N (Russian). Izv. Vyssk. Ucheb. Zaved, Khim. Khim. Technol. (Ivanovo) *12*:1483, 1969.

34. Bhura, D. C., and S. G. Tandon. Unsaturated N-Arylhydroxyamic Acids as Colorimetric Reagents for Vanadium: Spectrophotometric Determination With N-Phenyl-3-Styrylacrylhydroxyamic Acid. Anal. Chim. Acta. *53*:379, 1971.

35. Pilz, W., S. Komischke, and G. Prior. The Determination of Vanadium in Aqueous Solution, Air and Biological Material. Int. J. Environ. Anal. Chem. *1*:47, 1971.

36. Pilz, W., and S. Komischke. The Determination of Vanadium in Biological Material and Air. Int. J. Environ. Anal. Chem. *1*:275, 1972.

37. Ishii, H., and H. Einaga. Use of Calcichrome as a Spectrophotometric Reagent; X. The Vanadium IV and V Complexes of Calcichrome and a Spectrophotometric Method Based on the Vanadium IV Complex. Anal. Abstr. *21*:3355, 1971.

38. Chan, K. M., and J. P. Riley. The Determination of Vanadium in Sea and Natural Waters, Biological Materials and Silicate Sediments and Rocks. Anal. Chim. Acta. *34*:337, 1966.

39. Talvitie, N. A. Colorimetric Determination of Vanadium with 8-Quinolinol; Application to Biological Materials. Anal. Chem. *25*:604, 1953.

40. Laamanen, A., A. Lofgren, and L. Noro. Vanadium — A Specific Pollutant in the Air of Helsinki. Institute of Occupational Health. (Helsinki). Report No. 40. 1966.

41. Watanabe, H., H. Murayama, and S. Yamaoka. Clinical Findings in Vanadium Workers. Jap. J. Ind. Health. *8*:23, 1966.

42. Trace Characterization, Chemical and Physical. *In*: First Materials Research Symposium, Meinke, W. and B. Schribner (eds.), National Bureau of Standards. Monograph 100, Washington, D. C., 1967. 580 p.

43. West, P. W., and A. K. Mukherji. Separation and Microidentification of Metallic Ions by Solvent Extraction and Ring Oven Techniques. Anal. Chem. *31*:947, 1959.

44. Patrovsky, V. 2,3-Dihydroxynaphthalin als neues Reagens zur Extraktiven Photometrischen Bestimmung von Eisen-, Vanadin-, Titan, und Molybdanspuren (2,3 Dihydroxynapthal as a New Reagent for Extraction-Protometric Determination of Iron, Vanadium, Titanium, and Molybdenum Traces) Coll. Ect. Czech. Chem. Commun. (Prague) *35*:1599-1604, 1970.

45. Ghosh, N., and G. Siddhanta. Extraction-Photometric Determination of Vanadium (V) with N-(m-Nitrobenzoyl)-N-phenylhydroxylamine. Fresenius Z. Anal. Chem. (Wiesbaden) *253*:207, 1971.

46. Christian, G. D. Catalytic Determination of Vanadium in Blood and Urine. Anal. Letters. *4*:187, 1971.

47. Pearse, G. A., Jr. Spectrophotometric Determination of Vanadium with 2,6-Pyridinedicarboxylic Acid. Anal. Chem. *34*:537, 1962.

48. Siroki, M., and C. Djordjevic. Spectrophotometric Determination of Vanadium With 4-(2-Pyridylazo) resorcinol by Extracting of Tetraphenyl-Phosphonium and Arsonium Salts. Anal. Chim. Acta. 57:301, 1971.

49. Jungreis, E., and P. W. West. Specific Microdetermination of Vanadium (V) on the Ring Oven. Anal. Chim. Acta. *44*:440, 1969.

50. Majumdar, A. K., and S. K. Bhowal. Spectrophotometric Determination of Vanadium with N-Benzoyl-o-tolylhydroxylamine. Analyst. *96*:127, 1971.

51. Kuzmicheva, M. N. The Determination of Vanadium in Air. Gig. Sanit. (Moscow). *31*:229, 1966.

52. Sherwood, R. M., and F. W. Chapman, Jr. New Techniques for Analyzing Mixtures of Trace Metals. Anal. Chem. 27:88, 1955.

53. Sandell, E. B. Colorimetric Determination of Traces of Metals. New York, Interscience Publishers, Inc., 1944.

54. Von Jerman, L., and V. Jettmar. Polarographische Bestimmung von Vanadin in der Luft von Arbeitsraumen (Polarographic Determination of Vanadium in Work Areas). Z. Hyg. (Leipzig) *14*:12, 1968.

55. Cassani, F. Determinazione Potenziometrica del Titanio e del Vanadio (Potentiometric Determination of Titanium and Vanadium) Chem. Ind. (Praque) *51*:1248, 1969.

56. Sierra, F., C. Sanchez-Pedrino, T. Perez-Riuz, and C. M. Lozano. Amperometric Determination of Vanadates (Spanish) An. Quim. (Madrid). *66*:479, 1970.

57. Singh, D. and S. Sharma. Amperometric Permanganometric Estimations at Low Concentrations in Stirred Solutions. Ind. J. Chem. *8*:192, 1970.

58. Kostromin, A. I., A. A. Akhmetov, and L. N. Orlova. Coulometric Determination of Manganese (II), Cesium (III), and Vanadium (IV). (Russian) Zh. Anal. Khim. (Moscow) *25*:195, 1970.

59. Rigdon, L. P., and J. E. Harrar. Determination of Vanadium by Controlled-Potential Coulometry. Anal. Chem. *41*:1673, 1969.

60. Rhodes, H. U. Analyses of Atmospheric Dust by Electron Optics. PHS Grant 2R01 AP 00372, active February 1, 1966, to January 31, 1970. Terminal Report (unpublished). 27 p.

61. Blosser, E. R. Identification and Estimation of Ions, Molecules and Compounds in Particulate Matter Collected from Ambient Air. U. S. Environmental Protection Agency, Research Triangle Park, N. C. APTD-0705, 1971. 77 p.

62. Lander, D. W., R. L. Steiner, D. H. Anderson, and R. L. Dehm. Spectrographic Determination of Elements in Airborne Dirt. Appl. Spectrosc. *25*:270, 1971.

63. Morrow, N. L., and R. S. Brief. Elemental Composition of Suspended Particulate Matter in Metropolitan New York. Environ. Sci. Technol. *5*:786, 1971.

64. Boumans, P. W. J. M. Theory of Spectrochemical Excitation. New York, Plenum Press, 1966. p. 203-208.

65. Sugimae, A., and T. Hasegawa. Vanadium Concentrations in Atmosphere. Environ. Sci. Technol. *7*:444, 1973.

66. Hasegawa, T., and A. Sugimae. Heavy Metals in Atmospheric Particulates. Jap. Anal. *20*:840, 1971.

67. Tani, M. Methods and Actual Techniques of Measuring Heavy Metals in the Environmental Atmosphere. Kogai to taisaku. *8*:551-556, 1972.

68. Tabor, E. C., and W. V. Warren. Distribution of Certain Metals in the Atmosphere of Some American Cities. A.M.A. Arch. Ind. Health. *17*:145, 1958.

69. Frigieri, P., R. Trucco, R. Anzani, and E. Caretta. Spectroscopic Analysis of Elements Present in Airborne Materials. Chim. Ind. *54*:3, 1972.

70. Loseke, W. A., D. R. Scott, D. C. Hemphill, L. E. Holboke, R. J. Thompson, L. J. Pranger, and S. J. Long. Analysis of National Air Surveillance Network Particulate Samples for Trace Elements by Emission Spectrometry. U. S. Environmental Protection Agency, Research Triangle Park, N. C. (Presented at American Chemical Society Meeting, Chicago, August 27, 1973.)

71. Koh, K. J., and D. E. Ryan. Fluorescence and Metallic Valency States; Part VI. Determination of Vanadium with Benzoic Acid. Anal. Chim. Acta. *57*:295, 1971.

72. Karayannis, N. M., and A. H. Corwin. Volatilization and Separations of Metal Acetylacetonates at 115°C by Hyperpressure Gas Chromatography. J. Chromatogr. Sci. *8*:251, 1970.

73. Moshier, R. W., and R. E. Sievers. Gas Chromatography of Metal Chelates. New York, Pergamon Press, 1965. 163 p.

74. Taylor, M. L. Gas-liquid Chromatography of Trace Elements. *In*: New Trace Elements in Nutrition, Mertz, W. and W. E. Cornatzer (eds.). New York, Marcel Dekker, 1971. p. 363-389.

75. Martens, C. S., J. J. Wesolowski, R. Kaifer, and W. John. Sources of Vanadium in Puerto Rican and San Francisco Bay Area Aerosols. Environ. Sci. Technol. *7*:817, 1973.

76. Bando, S., and T. Imahashi. Activation Analysis of Vanadium in Deposits and Airborne Particulates. Jap. Anal. *18*:1477, 1969.

77. Dams, R., J. A. Robbins, K. A. Rahn, and J. W. Winchester. Nondestructive Neutron Activation Analysis of Air Pollution Particulates. Anal. Chem. *42*:861, 1970.

78. McKown, D. M., D. H. Gray, M. E. Eichor, and J. R. Vogt. Neutron Activation Analysis in Environmental Chemistry. Amer. Lab. 7:39-44, July 1973.

79. Craft, C. North Carolina State University, Raleigh, N. C. Personal communication with C. Sawicki, Chemistry and Physics Laboratory, U. S. Environmental Protection Agency, Research Triangle Park, N. C., 1973.

80. Livingston, H. D., and H. Smith. Estimation of Vanadium in Biological Material by Neutron Activation Analysis. Anal. Chem. *37*:1285, 1965.

81. Rancitelli, L. A., J. A. Cooper, and R. W. Perkins. Multielement Characterization of Atmospheric Aerosols by Instrumental Neutron Activation Analysis and X-ray Fluorescence Analysis. U. S. Atomic Energy Commission, Richland, Wash. Contract AT(45-1)-1830. 1973.

82. Rahn, K., J. J. Wesolowski, W. John, and H. R. Ralston. Diurnal Variation of Aerosol Trace Element Concentrations in Livermore, California. J. Air Pollut. Contr. Ass. *21*:406, 1971.

83. Rahn, K. A., R. Dams, J. A. Robbins, and J. W. Winchester. Diurnal Variations of Aerosol Trace Element Concentrations as Determined by Nondestructive Neutron Activation Analysis. Atomic Environ. *5*:413, 1971.

84. Gordon, G. E., W. H. Zoller, E. S. Gladney, and A. G. Jones. Trace Elements in the Urban Atmosphere. Nuclear Methods in Environmental Research, Topical Meeting Abstract, 1971.

85. Bingham, R. A. and R. M. Elliot. Accuracy of Analysis by Electrical Detection in Spark Source Mass Spectrometry. Anal. Chem. *43*:43, 1971.

86. Gilfrich, J. V., P. G. Burkhalter, and L. S. Birks. X-ray Spectrometry for Particulate Air Pollution — A Quantitative Comparison of Techniques. Anal. Chem. *45*:2002, 1973.

87. Rhodes, J. R., A. H. Pradzynski, and C. B. Hunter. Energy Dispersive X-ray Fluorescence Analysis of Air Particulates in Texas. Environ. Sci. Technol. *6*:922, 1972.

88. Giaque, R. D., F. S. Goulding, J. M. Jaklevic, and R. H. Pehl. Trace Element Determination With Semiconductor Detector X-ray Spectrometers. Anal. Chem. *45*:671, 1973.

89. Goulding, F. S., and J. M. Jaklevic. Trace Element Analysis by X-ray Fluorescence. Lawrence Berkeley Laboratory, Berkeley, Calif., Report No. UCRL-20625, May 1971.

90. Goulding, F. S., and J. M. Jaklevic. X-ray Fluorescence Spectrometer for Airborne Particulate Monitoring. U. S. Environmental Protection Agency, Office of Research and Monitoring, Washington, D. C. Publication No. EPA-R2-73-182, April 1973. 70 p.

91. Bowman, H. R., J. G. Conway, and F. Asaro. Atmospheric Lead and Bromine Concentration in Berkeley, Calif. Environ. Sci. Technol. *6*:558, 1972.

92. Dittrich, T. R., and C. R. Cothern. Analysis of Trace Metal Particulates in Atmospheric Samples Using X-ray Fluorescence. J. Air Pollut. Contr. Ass. *21*:716, 1971.

93. Johansson, T. B., R. Akelsson, and S. A. E. Johansson. X-ray Analysis: Elemental Trace Analysis at the 10^{-12}g Level. Lund Institute of Technology, Lund, Sweden. Publication No. *LUNP 7109*, August 1971.

94. Watson, R. L., J. R. Sjurseth, and R. W. Howard. An Investigation of the Analytical Capabilities of X-ray Emission Induced by High Energy Alpha Particles. Nucl. Instrum. Methods. *93*:69, 1971.

95. Leroux, J., and M. Mahmud. Flexibility of X-ray Emission Spectrography As Adapted to Microanalysis of Air Pollutants. J. Air Pollut. Contr. Ass. *20*:402, 1970.

96. Greenfelt, P., A. Akerstrom, and C. Grosset. Determination of Filter-Collected Airborne Matter by X-ray Fluorescence. Atmos. Environ. *5*:1, 1971.

97. Luke, C. L., T. Y. Kometani, J. E. Kessler, T. C. Loomis, J. L. Bove, and B. Nathanson. X-ray Spectrometric Analysis of Air Pollution Dust. Environ. Sci. Technol. *6*:1105, 1972.

98. Cooper, J. Comparison of Particle and Photon Excited X-Ray Fluorescence Applied to Trace Element Measurements of Environmental Samples. Nucl. Instrum. Methods. *106*:525, 1973.

99. Goulden, P. D., P. Brooksbank, and J. F. Ryan. Automated Solvent Extraction for the Determination of Trace Metals in Water by AAS. Amer. Lab. *8*:10-17, August 1973.

100. Chau, Y. K., and K. Lum-Shue-Chan. Complex Extraction of Vanadium for Atomic Absorption Spectroscopy; Determination of Microgram Quantities of Vanadium in Lake Waters. Anal. Chim. Acta. *50*:201, 1970.

101. Fishman, M. J., and M. W. Skougstad. Catalytic Detection of Vanadium in Water. Anal. Chem. *36*:1643, 1964.

102. Linstedt, K., and P. Kruger. Determination of Vanadium in Natural Waters by Neutron Activation Analysis. Anal. Chem. *42*:113, 1970.

103. Connor, J., N. F. Shimp, and J. C. F. Techow. A Spectrographic Study of the Distribution of Trace Elements in Some Podzolic Soils. Soil Sci. *83*:65-73, 1957.

104. Methods of Analysis of the Association of Official Agricultural Chemists (6th ed.). Association of Official Agricultural Chemists, Washington, D. C., 1945.

105. Kemp, D. M., and A. A. Smales. Determination of Vanadium in Rocks and Meteorites by Neutron Activation Analysis. Anal. Chim. Acta. *23*:297-410, 1960.

106. Shimp, N. F., J. Connor, A. L. Prince, and F. E. Bear. Spectrochemical Analysis of Soils and Biological Materials. Soil Sci. *83*:51-64, 1957.

107. Vanadium. National Academy of Sciences, Committee on Biological Effects of Atmospheric Pollutants, Washington, D. C., 1974.

108. Goeke, R. Determination of Vanadium in Ore Samples. Talanta. *15*:871-873, 1968.

109. Hammer, D. I., J. Finklea, R. H. Hendricks, T. A. Hinners, W. B. Riggan, and C. M. Shy. Trace Metals in Human Hair as a Simple Epidemiological Monitor of Environmental Exposure. *In*: Trace Substances in Environmental Health Symposium, Hemphill, D.D. (ed.). U. of Missouri, Columbia, Mo., 1971.

110. Colucci, A., P. Brubaker, J. Bumgarner, J. Creason, E. Faeder, J. French, D. Hammer, T. Hinner, D. Hinton, G. Love, C. Pinkerton, C. Sharp, W. Sovocool, W. Terrill, and M. Waters. Collection of Autopsy Tissue Sets — CHESS Pollutant Burden Protocol. In-house report, U. S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N. C., February 1973.

111. Gordos, A. A. U. of Michigan, Ann Arbor, Mich. Verbal communication on results of hair as an indicator of trace metal body burden with D. Worf, Human Studies Laboratory, U. S. Environmental Protection Agency, Research Triangle Park, N. C., June 1973.

112. Worf, D., A. Colucci, and C. Shy. National Tissue Specimen Bank. In-house report, U. S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N. C., 1973.

113. Finklea, J. F., D. Hammer, T. Hinners, and C. Pinkerton. Human Pollutant Burdens. U. S. Environmental Protection Agency, Research Triangle Park, N. C. (Presented at American Chemical Society Symposium, Los Angeles, April 1-2, 1971.)

114. Colucci, A., P. Brubaker, J. Bumgarner, J. Creason, E. Faeder, J. French, D. Hammer, T. Hinner, D. Hinton, C. Love, C. Pinkerton, C. Sharp, W. Sovocool, W. Terrill, and M. Waters. Collection of Maternal-Fetal Tissue Sets, CHESS Pollutant Burden Protocol. In-house report, U. S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N. C., February, 1973.

115. Trace Metal Analysis of Maternal-Fetal Tissue Sets, Human Scalp Hair, and Housedust. Stewart Labs, Inc., Knoxville, Tenn. EPA Contract No. 68-02-0663, December 1972.

116. Schroeder, H. A. Air Quality Monographs, Monograph No. 70-13, Vanadium. American Petroleum Institute, Washington, D. C., 1970. 32 p.

117. Soremark, R. Vanadium in Some Biological Specimens. J. Nutr. *92*:183-190, 1967.

118. Smith, W. H. Metal Contamination of Urban Woody Plants. Environ. Sci. Technol. *7*:631, 1973.

119. Zweibaum, F., and J. Moorhead. A Multi-element Atomic Absorption Analyzer. Atomic Absorption Newsletter. *6*(6):137, 1967.

120. Air Pollution Measurements of the National Air Sampling Network, Analyses of Suspended Particulates, 1957-1961. U. S. Department of Health, Education, and Welfare, Public Health Service, Washington, D. C. Public Health Service Publication No. 968, 1962.

121. Air Quality Data from the National Air Surveillance Networks and Contributing State and Local Networks, 1966 Edition. U. S. Department of Health, Education, and Welfare, Public Health Service, National Air Pollution Control Administration, Durham, N. C. NAPCA Publication No. APTD 68-9, 1968.

5. ENVIRONMENTAL APPRAISAL

5.1 OCCURRENCE

5.1.1. Natural Sources

Vanadium is one of the more abundant trace elements. It is found in igneous rocks, shales, iron meteorites and silicate meteorites, some phosphate deposits, uranium ore, and asphaltic deposits on all continents.[1,2] It resembles phosphorus and titanium and, like these two elements, is concentrated in basic rocks. Titanium magnetites are high in vanadium. In general, vanadium concentrations vary according to the type of rock or soil and are highest in shales and clays.[3] Vanadium occurs in the earth's crust as relatively insoluble salts, where it is commonly found in the trivalent state. The geochemical as well as the biochemical behavior of vanadium is determined by the fact that it may exist in three stable oxidation states: tri-, tetra-, and pentavalent forms.[1] The natural sources of airborne vanadium are believed to be marine aerosols and continental dust. Volcanic action might also be a source, but its contribution is believed to be small.

5.1.2. Human Uses

Sources of vanadium emissions can be placed into two major categories: those processes that use vanadium and its compounds as raw materials, and combustion processes with vanadium-containing fuel.

Combustion sources are by far the largest and the most widespread sources of vanadium air pollution, although other sources may have important local or regional significance. Coal and oil both contain varying amounts of vanadium, and their combustion resulted in estimated emissions of 17,000 megagrams (Mg) [metric tons (MT)] of vanadium into the air in 1968 (Table 5.1).[4]

5.1.2.1. Fuels — Crude oils vary greatly in their vanadium content. During the refining process, nearly all of the vanadium present remains in the residual fractions. Thus, kerosene, gasoline, diesel fuel, and home heating fuels contain relatively little of the metal, and the residual oils, which are employed in large combustion units, contain nearly all of the vanadium originally present in the crude oil. Information on the chemical form of airborne vanadium from oil combustion is limited. Bowden et al.[5] have reported that fly ash from residual fuel oil combustion may contain a variety of vanadium oxides such as V_2O_3, V_2O_4, or V_2O_5; and double metal oxides such as $Na_2O \cdot V_2O_5$ and $Fe_2O_3 \cdot V_2O_5$. However, V_2O_5 is thought to be the prevalent oxide of vanadium emitted into the atmosphere.[6]

Table 5.1. EMISSIONS OF VANADIUM BY SOURCE, 1968[4]

Source	Emissions, MT (Mg)	
	Subtotal	Total
Mining and processing	—	73
Metallurgical processing	—	131
Reprocessing	—	222
Steel:		
Blast furnace	57	—
Open-hearth furnace	151	—
Basic oxygen furnace	6	—
Electric arc furnace	N[a]	—
Cast iron	1	—
Nonferrous alloys	3	—
Chemicals and ceramics:		
Catalysts	2	—
Glass and ceramics	N[a]	—
Miscellaneous	2	—
Consumptive uses	—	17,006
Coal	1,587	—
Oil	15,419	—
Total	—	17,432

[a]N-negligible (less than 1 MT (Mg).

Power plants and utilities that burn residual and crude oil and coal and petroleum refineries that burn crude oil or residuals emit vanadium compounds as inorganic particulate and possibly as organocomplexes. The concentration of vanadium in fly ash collected downstream of the control device in two coal-fired power plants was reported[7] as 230 to 390 $\mu g/m^3$ and 1,350 to 1,580 $\mu g/m^3$.

Of the approximately 17,000 MT of vanadium emitted to the atmosphere in the United States during 1968 (about 0.28 percent of the total particulate matter emitted), over 80 percent came from the combustion of residual fuels.[4] Because of regulations that limit the sulfur content of fuel oils, however, the vanadium emissions from this source are expected to decrease. The desulfurization process coincidentally reduces significantly vanadium and other metallic impurities in the fuel oil. Radford and Rigg[8] found that the vanadium content of residual fuel oil made from a representative Venezuelan crude was reduced from 218 to 105 to 59 $\mu g/g$ as the sulfur content was reduced from 2.6 to 1.0 to 0.5 percent. On this basis, it appears that there is an almost one-to-one relationship between the vanadium reduction and the degree of desulfurization. The vanadium content of fuel oil was variable, however, ranging from 0.1 to 500 $\mu g/g$ in 100 samples analyzed.[9]

U. S. coal generally contains less than 35 $\mu g/g$ of vanadium, with western coals containing about one-half that value. Consumption of coal, especially western coal, is expected to increase. As particulate control technology improves (see Section 7), the total amount of vanadium emitted to the atmosphere may or may not significantly change.

Emission concentrations are dependent on several factors, including vanadium content of raw material and/or fuel, process type, method of operation, and the efficiency of the particulate control system.

Recently, coal-fired power plants have been the subject of several material balance studies for trace metals. One of the most thorough material balance studies was done on the Allen Steam Plant at Memphis, Tennessee, a 240-megawatt unit operated at 80 percent of full load.[10] Based on the results from the electrostatic precipitator inlet and outlet, the efficiency for vanadium removal in the plant is 99 percent (Table 5.2).

Table 5.2. MATERIAL BALANCE FOR VANADIUM AT A COAL-FIRED STEAM POWER PLANT[10]

	Vanadium collected, g/min[a]				
			In fly ash		
Run No.	In coal	In slag tank solids	At precipitator inlet	At precipitator outlet	Precipitator efficiency, %
5	26	13	14	—	—
7	87	57	53	0.75	99
9	26	14	9.7	0.12	99

[a] Samples collected isokinetically using an alundum filter followed by a millipore filter, and analyzed by instrumental neutron activation.

Considerable research is underway to determine trace element concentration by particle size. Analysis of fly ash from three coal-fired power plants indicates that much of the vanadium occurs in the small particle sizes (Table 5.3).[11] This concentration of vanadium in fine particles may be significant. Even though an electrostatic precipitator may have an overall 99 percent removal efficiency, the removal efficiency for small particles such as 1.3 μm and smaller will be less. It is these particles that are respirable and therefore are of greatest concern as a possible health hazard.

Table 5.3. VANADIUM CONCENTRATION OF COAL FLY ASH, BY PARTICLE SIZE[11,a]

	Concentration, μg/g		
Particle size, μm	Source A	Source B	Source C
1.3	345	260	183
2.0	277	382	182
4.6	240	265	282
8.5	201	193	204
13.	207	188	154
22.	199	193	136
33.	197	181	135
>33.	193	157	138

[a] Fly ash sized using a Bahco classifier and analyzed by neutron activation.

As part of EPA's Nationwide Fuel Surveillance Network, gasoline is collected from the 10 EPA Regions and analyzed for approximately 25 trace elements, including vanadium. Table 5.4 summarizes vanadium concentrations found in gasolines collected in the spring of 1972 from retail service station pumps.[12]

Table 5.4 also summarizes the vanadium content of selected residual fuel oils and crude oils.[13] One type of fuel oil, residual fuel oil No. 6, is the common fuel oil used for electric power generation

Table 5.4. VANADIUM CONCENTRATION IN GASOLINE, FUEL ADDITIVES, MOTOR OIL, FUEL OIL, CRUDE OIL, AND COAL[12-17]

Source	Number of samples	Average concentration	Range
Gasoline[a]			
Premium	22	0.001 μg/ml	0.001 to 0.002 μg/ml
Regular	22	<0.007 μg/ml	—
Low lead	6	<0.003 μg/ml	—
Fuel additives[b]	18	—	<0.001 to 0.031 μg/ml
Motor oil[c]	4	—	<0.001 to 0.007 μg/ml
Residual fuel oil[d]	20	67 μg/g	0.38 to 230 μg/g
Crude oil[d]	20	25 μg/g	<0.02 to 140 μg/g
Coal[e]	24	33 μg/g	10 to 62 μg/g

[a] Collected in spring, 1972, from retail service station pumps.

[b] Neutron-activation analysis. Fuel additive samples included: gas treatment, fuel-mix tune-up, engine tune-up, gas power booster, gasoline antifreeze, gas booster, and carburetor tune-up.

[c] Neutron-activation analysis.

[d] Neutron-activation analysis. Samples represent 20 different oil fields, both domestic and foreign. The residual fuel oil and crude oil are not necessarily from the same oil fields but represent 20 oil fields for each.

[e] Coal samples from Illinois, West Virginia, and Utah. Each coal was analyzed by X-ray fluorescence, optical emission-direct reading, and optical emission-photography; values from these three methods were then used to determine the average and range.

plants and large industrial boilers. Crude oil is of concern since several power plants have shifted from burning residual fuel oil to burning crude oil because of the increasing cost of low-sulfur residual fuel oil.[14] As of February 1972, three East Coast utilities were burning a total of 5,400 m^3/day of crude oil. By 1975, crude consumption is expected to climb to 28,000 m^3/day and will involve at least nine utilities.[14]

The vanadium concentrations for coals mined in various areas of the United States are summarized in Table 5.4 as well.[15]

5.1.2.2. Fuel Additives—In anticipation of regulations pursuant to Section 211 of the 1970 Clean Air Act Amendments on registration of fuels and fuel additives, EPA is analyzing consumer-purchased fuel additives for trace element content. As the first step of this surveillance effort, fuel additives were purchased from retail stores in the Research Triangle Park, N. C., area and analyzed for trace elements (Table 5.4)[16]

As part of EPA's Nationwide Fuel Surveillance Network, crankcase lubricating oils are also being collected for trace element analysis. Initial results for vanadium are shown in Table 5.4 for several grades of one brand of motor oil.[17]

5.1.2.3. Metallurgical Uses—The iron and steel industry has historically used the greatest tonnage of vanadium. All common wrought forms — sheet, wire, and tube — have been used. One of the commercial forms of vanadium that is added in steelmaking is ferrovanadium, an alloy consisting of iron and 50 to 80 percent vanadium. Ferrovanadium is brittle but not friable. It is not as corrosion-resistant as pure vanadium, but resists rusting in the normal open storage of a steel plant.

The ferrovanadium used for alloy additions in steelmaking is produced in electric arc furnaces. The charge consists of scrap steel, fused sodium metavanadate, carbon with silicon, and aluminum, or a combination of the two as a reducing agent. The furnace is not closed to the atmosphere. Most of the charge exists as metal slag or carbonaceous gases, but a substantial amount evolves as fumes. Emissions of vanadium into the atmosphere from the production of ferrovanadium totaled 131 Mg (MT) during 1968. These emissions constitute a potentially serious problem in the vicinity of ferrovanadium-producing plants, of which there are few in the United States. The emissions were mostly in the form of particles ranging from 0.1 to 1 μm in size.[4]

A large part of all the steel made finds its way back to steel-melting operations as scrap. In the refining stage of such operations, the contained alloy metals are oxidized out of the liquid steel as completely as possible. Vanadium from scrap steel is oxidized and discharged partly to air, and partly into slag, which can be run off, granulated, and discarded with reasonable safety. Today, a very high proportion of scrap steel is recycled by way of the basic oxygen furnace (BOF), which refines steel with a high-pressure jet of pure oxygen. This injection produces large amounts of fume that consist essentially of metallic oxides, which are normally collected by electrostatic precipitators. In recycling of steel in this manner, vanadium accounted for an estimated 0.02 percent of the estimated 21 kg (46 lb) of particulate emitted from the BOF per ton of steel produced, with a 97 percent emission control. During 1968, the BOF was used to produce 43.5 million Mg (MT) of steel.[4] Based on these production figures, an estimated 6.0 Mg (MT) of vanadium escaped into the atmosphere. This estimate was calculated using an emission factor of 21 kg (46 lb) of particulate emitted per ton of steel produced; 9×10^{-5} kg V/kg of particulate emitted (2×10^{-4} lb V/lb); and 97 percent emission control.

The site of particle deposition will depend largely on height of discharge and size of the particles. Atmospheric concentrations of vanadium at the boundary of a steel plant averaged 0.072 μg/m³ (Table 5.5).[18] Additional data from this same plant indicate that its particulate control equipment is 99.5 percent efficient,[19] and the plant would therefore produce lower emissions than those indicated in the earlier calculations and in Table 5.1.

Table 5.5. ATMOSPHERIC CONCENTRATIONS OF VANADIUM AT BOUNDARY OF TYPICAL STEEL PLANT, 1967[18,a]

Date of collection	Concentration, μg/m³
Jan. 1	0.054
Feb. 1	0.063
Feb. 22	0.040
Apr. 30	0.090
May 16	0.082
May 31	0.105
June 7	0.098
June 29	0.040
Aug. 3	0.040
Aug. 18	0.107
Sept. 8	0.092
Sept. 21	0.081
Oct. 5	0.040
Average	0.072
Range	0.040 to 0.107

[a] All samples were collected at the plant boundary in a community, but at an elevation of about 15 m. The Staplex high-volume air sampler with 8-μg, 25-cm glass fiber filters was used for the sample collection. Air concentrations were calculated as vanadium rather than vanadium pentoxide.

The high-cost tool and die steels are produced largely in vacuum-induction and vacuum arc-melting systems, in which vanadium carbides are added during the normal melting in a vacuum with cold-mold arc-fusion furances. Because of the melting system is closed, no vanadium should be released in any form to the atmosphere. The vanadium carbides are produced by carbothermic reduction of sodium metavanadate in a closed vacuum system operation, and a negligible amount of vanadium escapes into the atmosphere.

Vanadium is added to numerous steels in quantities from 0.2 to 4.5 percent (Table 5.6). Other traditional uses of vanadium in alloys are in welding rods, hard-facing alloys, magnetic alloys, high-temperature alloys, wear-resistant alloys, and steel alloys used in automotive parts (bolts, springs, engine valves, steam pipes and headers, and rotors). Good structural strength and a low-fission cross section make vanadium useful in nuclear applications; it is applied as the basic alloy in fuel cladding for advanced liquid-metal-cooled fast-breeder reactor.[8] Consumption of vanadium by end product is presented in Table 5·7.

Table 5.6. STEELS CONTAINING VANADIUM[3]

Type of steel	Vanadium content, %
Tool and die	0.2 to 4
High-speed tool	1 to 4
Cold	0.2 to 4.5
High carbon, high chromium	1 to 4
Hot	0.3 to 0.5
Stainless, Type 422	0.3
Carbon-vanadium	0.1 to 0.2
Chromium-vanadium	0.05 to 0.5
Manganese-vanadium	0.05 to 0.2
Vanadium-spring steels	0.08 to 0.2

Table 5.7. METALLURGICAL USES OF VANADIUM IN THE UNITED STATES[20]
(Mg (MT) of contained vanadium)

End use	1967	1968
Steel (ingots and castings):		
High-speed and tool	678	553
Stainless	35	45
Alloy (excluding stainless and tool)	1,910	2,350
Carbon	746	990
Other steel	5	6
Cast irons	49	52
Cutting and wear-resistant materials	12	15
Welding and hardfacing rods and materials	11	11
Magnetic alloys	4	5
Nonferrous alloys[a]	557	416

[a] Principally titanium-base alloys.

Two relatively young segments of the steel industry — high-strength, low-alloy steels (HSLA) and continuous casting of steel slabs and billets — are expected to exceed all other cumulative uses of vanadium.[3] Vanadium in HSLA steels increases strength by substantially increasing the yield point. The amount of vanadium used (approximately 0.1 percent) and the techniques employed are such that other properties of the steels, particularly ductility and weldability, are not adversely affected.[3] Aluminum has been used extensively as a deoxidant in slab casting and as a grain refiner in the continuous casting of billets, but vanadium has been substituted to avoid nozzle clogging of the tundish (caster feed pot) by alumina formed during use. Vanadium increases yield strength, increases resistance to hot-tearing, and provides a cleaner steel surface. However, impact strength is reduced somewhat, and a higher yield point makes bending more difficult.

Apart from its use in steels, vanadium is a major alloying element in high-strength titanium alloys. Aluminothermic vanadium, an alloy of aluminum and vanadium (4 percent), is added to titanitum as a master alloy. The effect of vanadium on titanium is strong beta stabilization, which promotes good hot and cold workability.[8] Because of their reactive nature, these alloys are melted under vacuum in a closed system. During the processing of steel ingots to wrought shapes, the steel is oxidized superficially, and thick oxide or mill scale is formed. Various grinding and machining operations produce comminuted materials, most of which are not recycled; but the materials themselves are quite inert and do not become airborne. In preparation for cold finishing, steels are pickled in baths of strong acids to remove surface defects. The spent acids contain a considerable volume of metal, along with a residue of sludge that is rich in halide salts of vanadium. Unless treated properly, the large quantities of spent acids could be a potential source of pollution.

The many shapes in which common ferrous alloys containing vanadium are fabricated and used require chemical treatment (such as pickling of scale) and high-temperature operations (such as welding, flame cutting, and hot working). Four vanadium metal operations — furnace addition of vanadium, tap of a furnace heat, oxyacetylene cutting, and arc-welding beams — would be expected to produce the greatest amount of fume. Data from a steel plant show a minimum of exposure of vanadium (Table 5.8).[18] The coating on welding rods is considered a potential source of vanadium fume, although little information exists on the toxic substances present as impurities or on their concentrations in arc-welding fumes.

5.1.2.4. Chemical Uses — The following are some of the many unique uses of vanadium compounds in the area of chemistry:

- Manufacture of sulfuric acid.
- Manufacture of phthalic anhydride.
- Manufacture of maleic anhydride.
- Manufacture of aniline black.
- Oxidation of cyclohexane to adipic acid.
- Oxidation of ethylene to acetaldehyde.
- Oxidation of anthracene to anthraquinone.
- Oxidation of toluene or xylene to aromatic acids.
- Oxidation of furfural to fumaric acid.
- Oxidation of hydroquinone to quinone.
- Oxidation of butene-2 and 1,3-butadiene to maleic anhydride.
- Ammonolysis oxidation of toluene, *m*-xylene, *p*-xylene, and propylene.
- Manufacture of vinyl acetate from ethylene.
- Manufacture of cyclohexylamine from cyclohexanol and ammonia.
- Catalytic combustion of exhaust gases.
- Catalytic synthesis of ethylene-propylene rubber.

Table 5.8. CONCENTRATIONS OF VANADIUM PENTOXIDE IN AIR AT POINT SOURCES AT A STEEL PLANT[a]

Location	Operation	Vanadium content of steel, %	V_2O_5 concentration in breathing zone, mg/m^3 of air[a]
V-ton furnace	Furnace addition of vanadium	0.15 to 0.25	0.006
		0.15 to 0.25	0.007
		0.15 to 0.25	0.078
		0.15 to 0.25	0.007
		1.8 to 2.1	0.019
		1.8 to 2.1	0.013
V-ton furnace	Tap of furnace heat	0.5 to 0.7	0.004
		0.5 to 0.7	0.011
		0.5 to 0.7	0.004
		0.5 to 0.7	0.02
Press forge	Oxyacetylene cutting of forging	1.02	0.002
		1.05	0.008
		0.15 to 0.4	0.004
		0.15 to 0.4	0.015
		0.15 to 0.4	0.003
		0.15 to 0.4	0.011
		0.1 to 0.15	0.01
		0.1 to 0.15	0.002
		0.1 to 0.15	0.005
		0.1 to 0.15	0.001
Beam-yard weld bed	Arc-welding beams	0.8	0.003
		0.8	0.006
		0.8	0.002
		0.8	0.002
		0.8	0.004
		0.8	0.004
		0.8	0.003
		0.8	0.005
		0.8	0.004
		0.8	0.005

[a] Threshold limit values for vanadium fume as vanadium pentoxide: 0.05 mg/m^3, ceiling value, in 1972.[21]

Estimates of vanadium consumed by the chemical industry vary from 179 Mg (MT) in 1965 to 120 Mg (MT) in 1967, with projections of 182 to 227 Mg (MT) in 1976.[3] The more important uses are to catalyze the synthesis of sulfuric acid, the oxidation of hydrocarbons, and the polymerization of mono- and diolefins.[3]

The oxidation of sulfur dioxide to sulfur trioxide in the production of sulfuric acid consumes by far the greatest amount of vanadium — estimated at 69 Mg (MT) vanadium pentoxide in 1965.[3] Vanadium-based catalysts (such as vanadium pentoxide on asbestos) are reported to be the preferred catalysts for the reaction because of longer life, higher average efficiency, immunity to poisoning by arsenic, chlorine, etc., and greater physical ruggedness. A vanadium oxide catalyst is also used in two closely related processes — oxidation of aromatic hydrocarbons to phthalic anhydride, and oxidation of benzene to maleic anhydride.

Vanadium compounds other than vanadium pentoxide are also used. For example, ammonium metavanadate is mentioned in connection with the manufacture of adipic acid, vanadium oxytrichloride in connection with the production of ethylene-propylene synthetic rubber, and vanadium trichloride in stereo-specific catalyst systems and as an intermediate in making high-purity metals and intermetallics.[3]

In dye manufacture and dyeing, vanadium compounds are widely used in production of aniline black. Vanadium salts are added as catalysts to a mixture of aniline hydrochloride and potassium or sodium chlorate. Vanadium compounds are used as mordants in the dyeing and printing of cotton, and particularly for fixing aniline black on silk. Ammonium metavanadate has been used as a catalyst in the dyeing of leather and fur. Some modern quick-drying inks require addition of ammonium metavanadate.[3]

Generally, total consumption of vanadium in these chemical processes is small; furthermore, its principal use is as a catalyst, not as an ingredient. Potential hazards other than in point-source industrial operations appear limited. But these uses of vanadium do leave two aspects for consideration: first, vanadium compounds in the products will have very wide distribution; second, the spent catalyst will be disposed of in some manner. Some spent catalysts are being reprocessed to vanadium products; however, the extent of this recycling has not been determined.

Closely allied to the chemical industry is polymer synthesis and processing. Total consumption of vanadium in this industry, although small, is growing rapidly; 1975 levels may be 91 to 136 Mg (MT).[3] Polymer products such as packaging films are widely distributed, sometimes in connection with food, and large quantities are incinerated. Questions have been raised regarding vanadium extracted from polymers during food storage and from vanadium emission during incineration. Fortunately, plastics have been found to contain only 1 ng/g or less of vanadium, which is not considered hazardous.

5.1.2.5. Ceramics — The use of vanadium in the ceramic industry is on the decline. Vanadium compounds are not currently used in the manufacture of glass fibers, frits, or refractories. Ammonium metavanadate continues to play a primary role in various ceramic glazes, particularly the zircon vanadium blues. These glazes are not used in dinnerware, but in such applications as wall tile. Furthermore, the vanadium is tightly bound in substitutional solid solution in the zircon structure and is therefore very stable.

5.1.2.6. Electronics Industry — The growth of the electronics industry, particularly the proliferation of solid-state devices, has emphasized the role of a number of transition elements, including vanadium. Considerable research is underway, and it is likely that new applications will evolve. For example, the insulator-to-metal transition in vanadium tetroxide at about 60°C is intriguing; the resistance drops by several orders of magnitude over a very narrow temperature range.

5.2 CONCENTRATIONS

5.2.1. Air

Vanadium concentrations over relatively unpopulated areas of the earth range from 0.02 to 0.8 ng/m^3 in the eastern Pacific Ocean[22] and from 0.21 to 1.9 ng/m^3 in remote northwestern Canada.[23]

In rural areas of the United States, values ranged from 0 to 13 ng/m^3 for 1960 through 1965, and from 1.0 to 64.6 ng/m^3 for 1967.[2] Although very few studies have been specifically designed to determine ambient concentrations of vanadium, the element has been included in the trace metals analysis of several more general air pollution studies. For several years, NASN routinely reported the vanadium content in the particulate samples collected at its many monitoring sites across the country. In addition, vanadium concentrations were determined in several short-term monitoring projects aimed at establishing the overall air pollution pattern within a particular geographical area.

5.2.1.1. NASN Studies — Samples of suspended particulate matter collected at the NASN sites from 1957 to 1969 were analyzed for trace metals, including vanadium. The minimum level of detection for the spectrographic analytical method is considered to be 3 ng/m^3 for urban sites and 1 ng/m^3 for nonurban sites (located in national parks or other remote areas). Concentrations falling below these threshold levels were entered at one-half the minimum level of detection for purposes of computation. Additional statistics (maximum, minimum, etc.) are available for the year 1968, the only recent year in which the 24-hour particulate samples were individually analyzed. In other years, the individual samples were combined into quarterly composites.

5.2.1.1.1. Long-term trends — Table 5.9 presents the annual average vanadium concentration for urban NASN sites with complete data for the 5-year period of 1965 through 1969.[24] Table 5.10 presents similar data for the nonurban sites.

The overall averages do not seem to reflect a clear pattern of increasing vanadium concentrations over the period. Rather, it appears that there were considerable year-to-year fluctuations in ambient levels, with substantially higher levels in 1967 and 1969 than in the other 3 years. This observation seems consistent with the belief that the major source of vanadium emissions is the combustion of fuel oil for power production and space heating, which, though generally increasing, varies considerably from year to year in response to the severity of the winter season. Concentrations at the nonurban sites seem to follow the basic trend of the urban sites at approximately one-tenth the level.

Figures 5.1 and 5.2 present the annual averages for the 5-year period in the form of histograms for the urban and nonurban sites, respectively. Although the distribution remains fairly stable from year to year, in the high concentration year of 1969, much of the increase occurred at sites that had traditionally exhibited very low ambient levels.

5.2.1.1.2. Geographic distribution — Ambient concentrations of vanadium are by no means geographically uniform (see Figure 5.3, based on Tables 5.9 and 5.10). Sites that have experienced relatively high long-term vanadium concentrations occur in quite well-defined geographic clusters. The highly urbanized region along the Atlantic coast from New England to Virginia is the nation's leader in vanadium concentrations. In addition to being densely populated, this region is highly dependent on fuel oil (much of it from vanadium-rich crude oil of Venezuela) for power production, space heating, and industrial purposes.[2] Also, almost all of the plants producing vanadium chemicals in the United States are located within this region.[25]

Relatively high concentrations are also found in Puerto Rico and Hawaii (where fuel oil, rather than coal, is the major source of energy), in the Chicago metropolitan area, and in the urban centers along the Pacific Coast. The average vanadium levels throughout the remainder of the country were generally less than 10 ng/m^3 for the 5-year period. This undoubtedly reflects the availability and use of coal, natural gas, and vanadium-poor fuel oil in those areas.

Table 5.9. ANNUAL AVERAGE VANADIUM CONCENTRATIONS — NASN URBAN SITES, 1965-1969[74] (ng/m^3)

Site	Annual avg.					5-Yr. avg.
	65	66	67	68	69	
AZ, Phoenix	5	b[a]	b	b	8	4
Tucson	b	b	b	b	8	3
CA, Los Angeles	11	21	18	12	15	15
Oakland	19	14	29	12	30	21
San Diego	28	16	20	6	31	20
San Francisco	24	17	16	10	23	18
CN, New Haven	377	327	492	533	897	525
DE, Wilmington	149	115	189	236	372	212
DC, Washington	128	118	164	87	139	127
GA, Atlanta	b	14	4	b	23	9
HI, Honolulu	38	27	15	49	32	32
ID, Boise City	b	b	b	b	13	3
IL, Chicago	83	47	58	35	96	64
IN, Hammond	33	17	34	12	98	29
Indianapolis	13	9	15	3	22	10
South Bend	131	50	30	5	47	53
IA, Des Moines	4	b	b	b	12	4
KN, Wichita	b	b	b	b	9	3
KY, Louisville	7	b	9	b	17	7
LA, New Orleans	14	9	26	7	22	16
MD, Baltimore	71	254	199	185	279	198
MI, Detroit	11	5	11	6	19	10
MN, Minneapolis	9	5	6	2	17	8
St. Paul	9	5	24	8	24	14
MO, Kansas City	14	4	7	3	11	8
MT, Helena	b	b	b	b	b	b
NE, Omaha	3	b	b	b	7	3
NV, Las Vegas	b	b	b	b	7	b
NH, Concord	37	73	143	53	95	80
NJ, Glassboro	72	37	51	34	97	58
Jersey City	319	262	487	355	202	325
Newark	349	257	344	265	146	272
Perth Amboy	267	202	390	104	123	217
NM, Albuquerque	b	b	b	b	8	b
NC, Charlotte	23	19	18	14	38	22
OH, Okron	4	b	6	b	10	5
Cincinnati	8	b	8	b	12	6
Cleveland	4	b	4	b	14	5
Columbus	5	b	b	b	10	4
Toledo	7	b	5	b	7	4
Youngstown	3	4	4	b	9	4
OK, Oklahoma City	b	b	b	b	5	b
Tulsa	b	b	b	b	4	b
OR, Portland	46	30	37	41	54	42
PA, Philadelphia	398	133	263	358	434	317
Pittsburgh	b	b	16	3	15	7
Reading	87	92	81	96	141	99
Warminster	58	94	98	37	59	69
PR, Bayomon	41	62	131	78	70	76
Catano	105	67	55	80	212	104
RI, Providence	256	341	271	389	607	373
TN, Chattanooga	5	4	5	3	13	6
Memphis	b	b	b	b	7	b
TX, Houston	b	b	b	b	6	b
San Antonio	b	b	b	b	b	b
UT, Salt Lake City	7	b	4	b	b	3
VA, Norfolk	76	32	50	57	131	69
WA, Seattle	39	28	22	19	37	29
WV, Charleston	17	14	15	16	33	19
WI, Milwaukee	7	b	4	b	18	5
Overall avg.	51	48	65	54	81	60

[a] b—below minimum detectable level.

It is worth noting that even *nonurban* sites located along the eastern seaboard exhibit elevated vanadium concentrations, in marked contrast to the barely detectable levels observed at nonurban sites throughout the rest of the country. This suggests that urban emisions of vanadium in the Northeast are adversely affecting the air quality in rural areas within the region.

Table 5.11 lists all urban sites that in 1968 exceeded an annual average vanadium concentration of 100 ng/m^3 and all nonurban sites that exceeded an annual average of 10 ng/m^3. All sites are located along the eastern seaboard, and in each case, the maximum vanadium concentrations occurred during the peak period of fuel oil consumption for space heating.

5.2.1.1.3. Seasonal distribution — From what has preceded, it seems reasonable to suspect that ambient concentrations of vanadium increase during the colder portion of the year as fuel oil consumption increases to meet the space-heating demand. To test this hypothesis more formally, average concentrations were calculated for the first and fourth quarters (combined) and the second and

Table 5.10 ANNUAL AVERAGE VANADIUM CONCENTRATIONS — NASN NONURBAN SITES, 1965 - 1969[24]
(ng/m^3)

Site	Annual avg.					5-yr avg.
	1965	1966	1967	1968	1969	
AZ, Grand Canyon N.P.[a]	1	b[b]	b	b	b	b
AR, Montgomery Co.	b	b	b	b	b	b
CA, Humboldt Co.	b	b	b	b	b	b
CO, Mesa Verde N.P.	b	b	b	b	b	b
IN, Parke Co.	1	1	b	b	2	b
ME, Acadia N.P.	8	11	29	4	17	13.8
MD, Calvert Co.	6	24	44	19	34	25.4
MO, Shannon Co.	b	b	1	b	b	b
MT, Glacier N.P.	b	b	b	b	b	b
NE, Thomas Co.	b	b	b	b	b	b
NV, White Pine Co.	2	b	b	b	b	b
NH, Coos Co.	2	9	7	5	12	7.0
NY, Jefferson Co.	5	6	9	5	17	8.4
NC, Cape Hatteras	2	5	5	4	8	4.8
OK, Cherokee Co.	b	b	b	b	b	b
OR, Curry Co.	b	b	b	b	b	b
PA, Clarion Co.	1	1	1	b	3	1.2
RI, Washington Co.	24	31	48	33	46	36.4
SC, Richland Co.	1	2	4	2	6	3.0
SD, Black Hills	b	b	3	b	1	b
TX, Matagorda Co.	1	b	b	b	2	b
VT, Orange Co.	13	28	64	41	53	39.8
VA, Shenandoah N.P.	1	1	2	1	8	2.6
Overall average	3	5	9	5	9	6.3

[a] N.P. National Park.
[b] Below minimum detectable level.

third quarters (combined) for the year 1968 (Table 5.12). Nationwide, the average vanadium level in the colder half of the year was more than triple the level of the warmer 6 months. This strongly confirms the dominant role played by fuel oil combustion with regard to concentration of vanadium in ambient air.

5.2.1.1.4. Size distribution of particulate vanadium — The size distribution of vanadium-bearing particulate matter emitted to the atmosphere is of interest since it affects the proportion of the emitted particulate vanadium that will remain suspended and also the proportion that will be respirable by human beings. Size distribution of trace metals was the subject of a special NASN study[26] conducted during 1970 (Table 5.13). Approximately 80 percent of the suspended vanadium particulate has a mass median diameter (MMD) of less than 2 μm. This means that virtually all of the suspended vanadium particulate is below the upper limit of human respirability (generally considered to be 5 μm). Lead was the only other trace metal evaluated in the study that was associated with such small particles. The vanadium size distribution is quite uniform from site to site, further evidence that most airborne vanadium may be traced to a common source, fuel oil combustion.

5.2.1.2. Area Studies — Although no studies designed specifically to determine the influence of vanadium sources on ambient concentrations have been reported, the metal has been included among the trace metals determined in several special area studies of air pollution patterns.

Table 5.11 NASN URBAN SITES EXCEEDING 100 ng/m^3 AND NONURBAN SITES EXCEEDING 10 ng/m^3, 1968[74]

Site	Annual avg.	Maximum quarterly avg./first quarter of yr	Maximum 24-hr avg./date and month
Urban:			
Boston, MA	617	1,102	1,600 (2/28)
New Haven, CT	533	1,134	2,500 (1/29)
Providence, RI	389	944	1,600 (2/15)
Philadelphia, PA	358	846	1,600 (1/3)
Jersey City, NJ	355	994	2,300 (1/2)
Bayonne, NJ	285	731	930 (2/27)
Hartford, CT	269	661	1,700 (1/3)
Newark, NJ	265	649	1,400 (2/27)
Wilmington, DE	238	591	1,100 (2/18)
Camden, NJ	215	408	770 (2/26)
Paterson, NJ	210	651	1,400 (2/27)
Worchester, MA	198	523	950 (2/5)
Baltimore, MD	185	530	720 (2/27)
E. Providence, RI	126	226	560 (2/27)
Harrisburg, PA	105	288	940 (1/28)
Perth Amboy, NJ	104	247	450 (1/3)
Nonurban:			
Orange Co., VT	41	96	180 (1/29)
Washington Co., RI	33	46	150 (4/12)
Calvert Co., MD	19	36	55 (3/26)
Kent Co., DE	18	39	130 (1/3)

5.2.1.2.1. Kanawha Valley — During 1964-1965, a comprehensive air pollution study was conducted in the Kanawha Valley of West Virginia.[27] Twenty-four-hour samples of suspended particulate matter were collected at 14 of the sampling sites (Table 5.14 and Figure 5.4). Samples from selected sites were composited on a seasonal basis and analyzed for trace metal content by the NASN emission spectrographic method. In addition, monthly dustfall samples were collected at several of the sampling sites.

Vanadium concentrations in the study are quite low (Table 5.14), averaging about one-tenth the national average for urban areas. This is probably due to the abundance of coal and natural gas in the area, which tends to minimize the use of fuel oil. (Both of the major power plants in the valley are coal-fired, and an estimated 95 percent of the households are heated with natural gas). The data do indicate, however, that maximum levels tend to occur during the fall and winter seasons and in the areas of highest population density.

5.2.1.2.2. Birmingham — During 1964-1965, seasonal levels of trace metals were determined for suspended particulate samples collected at 10 Birmingham, Alabama, area sampling sites (Table 5.15).[28] In Birmingham, as in West Virigina, the vanadium levels were quite low. Here, the familiar seasonal pattern was not in evidence, and levels appeared fairly uniform throughout the study area.

5.2.1.2.3. Chicago — Neutron-activation analysis was used to determine the trace metal content of 24-hour samples of particulate matter collected on a single day (April 4, 1968) at 22 sampling stations distributed over the Chicago metropolitan area (Figure 5.5).[29] The vanadium concentra-

Table 5.12. VANADIUM CONCENTRATIONS BY SEASON, 1968[24]
(ng/m³)

Site[a]	First and fourth quarter	Second and third quarter	Ratio
CA, Burbank	19	2	9.5
Long Beach	18	2	9.0
Oakland	18	6	3.0
San Diego	11	2	5.5
CN, Hartford	449	74	6.1
New Haven	867	186	4.7
DE, Kent Co.	26	8	3.3
Newark	52	9	5.8
Wilmington	395	63	6.3
DC, Washington	133	41	3.2
FL, Jacksonville	51	19	2.7
Tampa	30	19	1.6
HI, Honolulu	86	6	14.3
IL, Chicago	62	8	7.8
IN, Hammond	18	7	2.6
South Bend	6	6	1.0
LA, New Orleans	9	5	1.8
ME, Acadia Nat'l Park	2	5	0.4
MD, Baltimore	263	100	2.6
Calvert Co.	24	14	1.7
MA, Boston	845	347	2.4
Worchester	341	64	5.3
MI, Detroit	5	7	0.7
NV, Reno	24	14	1.7
NH, Concord	72	29	2.5
Coos Co.	6	5	1.2
NJ, Bayonne	432	147	2.9
Camden	285	137	2.1
Glassboro	44	24	1.8
Hamilton	102	47	2.2
Jersey City	547	122	4.5
Newark	391	106	3.7
Paterson	346	90	3.8
NJ, Perth Amboy	144	59	2.4
Trenton	64	59	1.1
NY, Jefferson Co.	8	2	4.0
NC, Cape Hatteras	5	4	1.3
Charlotte	23	4	5.8
Durham	6	3	2.0
OR, Portland	64	21	3.0
PA, Allentown	135	39	3.5
Bethlehem	83	30	2.8
Harrisburg	153	49	3.1
Johnstown	9	3	3.0
Philadelphia	552	142	3.9
Pittsburgh	5	2	2.5
Reading	130	66	2.0
Scranton	102	30	3.4
Warminster	58	11	5.3
West Chester	42	8	5.3
York	154	12	12.8
PR, Bayomon	92	63	1.5
Catano	84	76	1.1
Guayanillo	54	40	1.4
Ponce	12	9	1.3
RI, E. Providence	165	85	1.9
Providence	679	123	5.5
Washington Co.	37	30	1.2
SC, Columbia	12	2	6.0
Greenville	17	2	8.5
VT, Burlington	77	55	1.4
Orange Co.	71	9	7.9
VA, Hampton	43	17	2.5
Norfolk	77	35	2.2
Richmond	92	16	5.8
WA, Seattle	33	5	6.6
WV, Charleston	20	12	1.7
Overall	185	52	3.6

[a] Sites with more than one quarterly average below the minimum detectable level were not included in the analysis. The overall means differ significantly at the 99 percent confidence level (t-test).

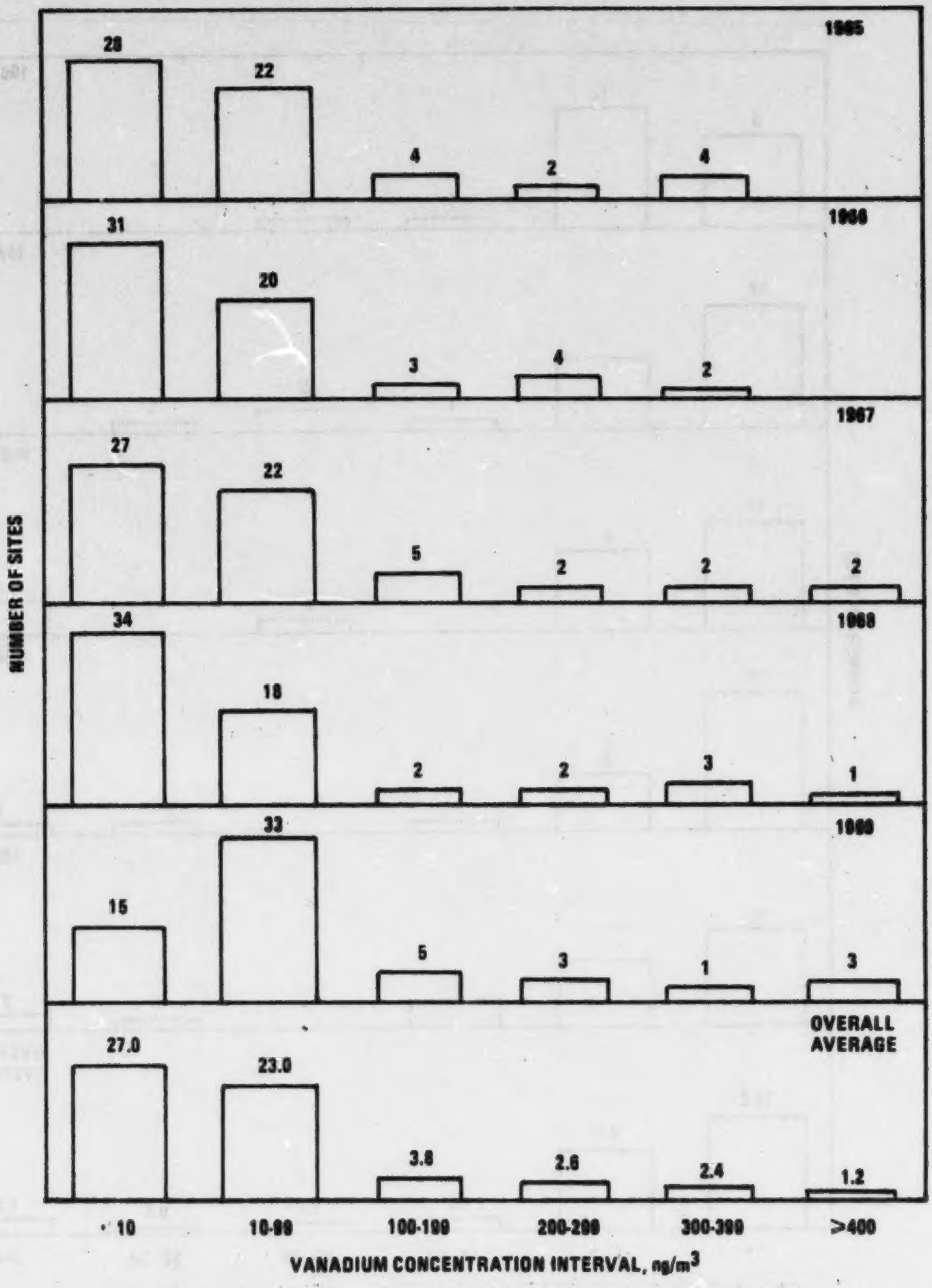


Figure 5.1. Histogram for NASN urban sites, 1965-1969.[24]

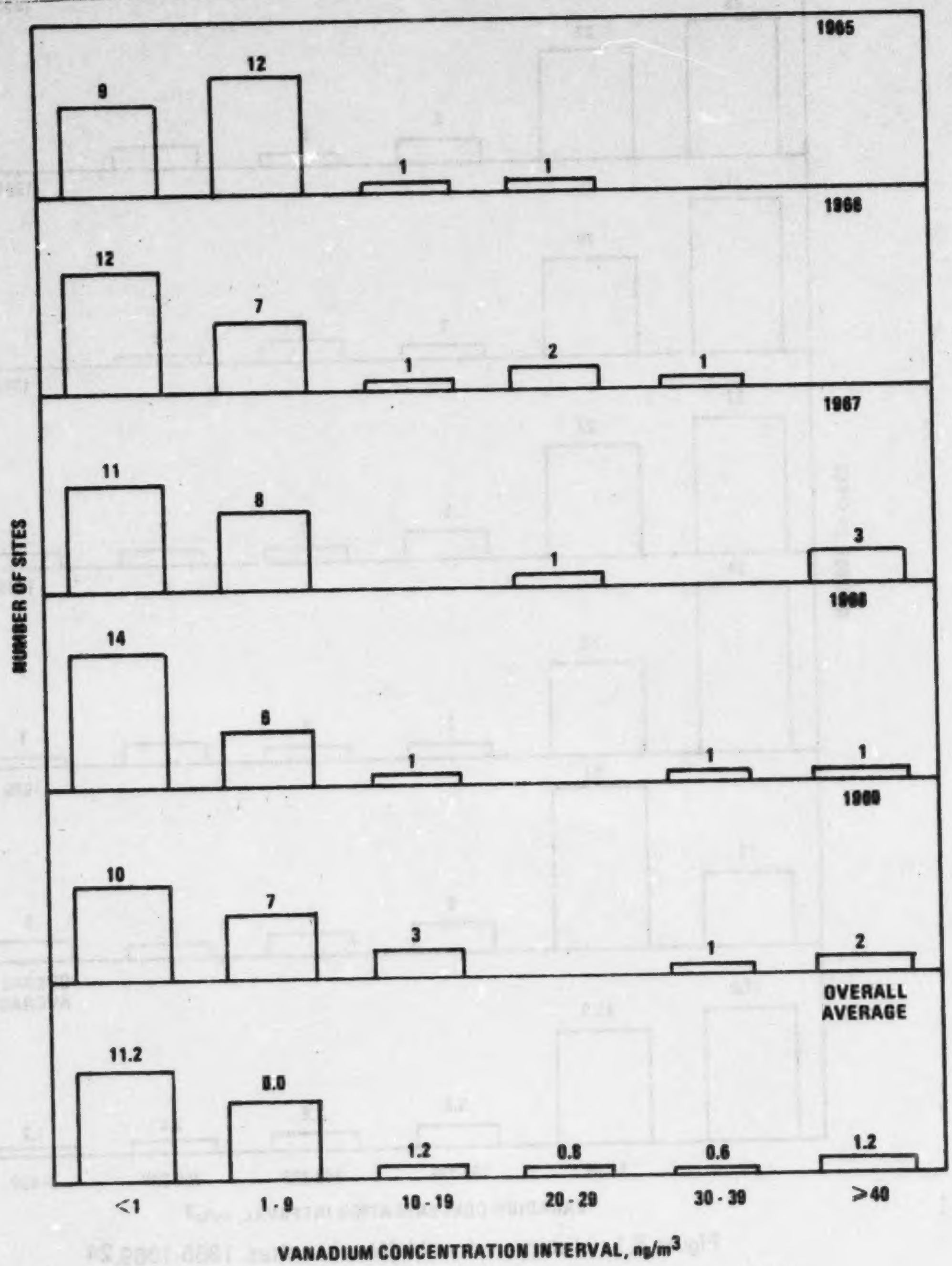


Figure 5.2. Histogram for NASN nonurban sites, 1965-1969.[24]

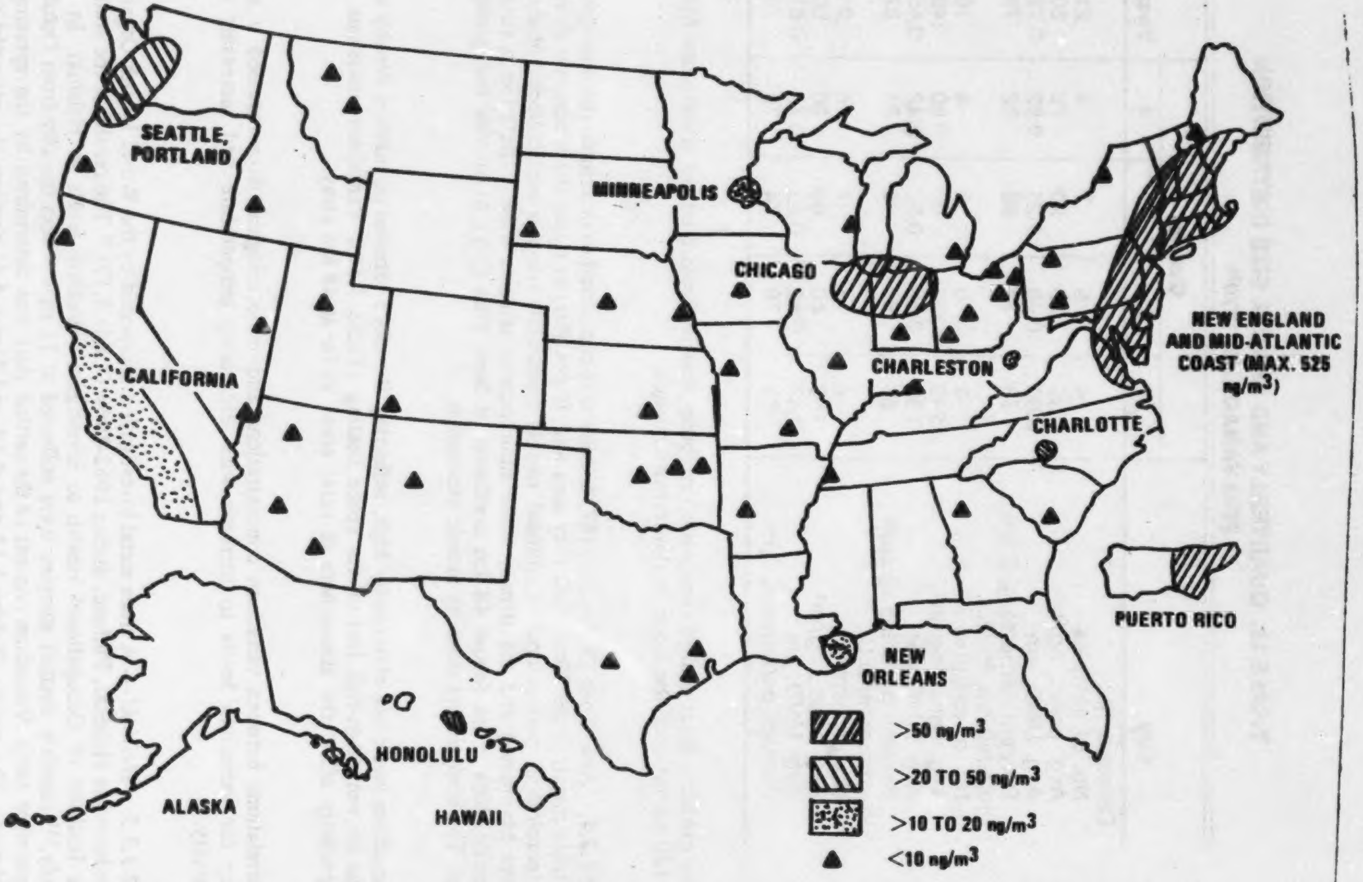

SEATTLE, PORTLAND
CALIFORNIA
MINNEAPOLIS
CHICAGO
CHARLESTON
CHARLOTTE
NEW ENGLAND AND MID-ATLANTIC COAST (MAX. 525 ng/m^3)
PUERTO RICO
NEW ORLEANS
ALASKA
HONOLULU
HAWAII
▨ >50 ng/m^3
▨ >20 TO 50 ng/m^3
▨ >10 TO 20 ng/m^3
▲ <10 ng/m^3

Figure 5.3. Geographic distribution of vanadium concentrations. Generalized on basis of 5-year (1965-1969) averages for NASN sites.[24]

Table 5.13. QUARTERLY AND ANNUAL SIZE DISTRIBUTION FOR VANADIUM, 1970[26]

City	Quarter 1	Quarter 2	Quarter 3	Quarter 4	Year
Chicago, Ill.:					
No. of samples	4	6	7	4	21
Avg. conc., ng/m^3	120	30	20	70	60
Avg. MMD, μm	0.66	0.99	1.25	0.52	0.72
Percent particles ⩽ 2 μm	76	74	69	82	76
Philadelphia, Penn.:					
No. of samples	1	6	7	4	18
Avg. conc., ng/m^3	240	100	90	120	140
Avg. MMD, μm	0.35	0.45	0.82	0.42	0.45
Percent particles ⩽ 2μm	83	82	78	84	82
Washington, D.C.:					
No. of samples	4	5	7	5	21
Avg. conc., ng/m^3	150	60	60	90	90
Avg. MMD, μm	0.47	0.62	0.88	0.75	0.61
Percent particles ⩽ 2μm	81	79	74	79	79

tions exhibit a great deal of site-to-site variability, ranging from 2 ng/m^3 at suburban Morton Grove to 120 ng/m^3 near the Loop in downtown Chicago.

5.2.1.2.4. New York City — In 1968, a study was conducted to determine the atmospheric burden of trace metals in the New York City area and, if possible, to relate these concentrations to certain meteorological parameters.[30] Continual weekly particulate samples were collected at sites located at lower Manhattan and the Bronx, while simultaneous samples were collected in a rural area near Tuxedo, New York (about 48 km northwest of New York City), to provide background information. The analysis was done by atomic absorption.

Vanadium levels are abnormally high, reflecting the city's extreme population density and dependence on vanadium-rich fuel oil for space heating (Table 5.16). Vanadium emissions in the city apparently affect the atmosphere of rural areas as far as 48 km away.

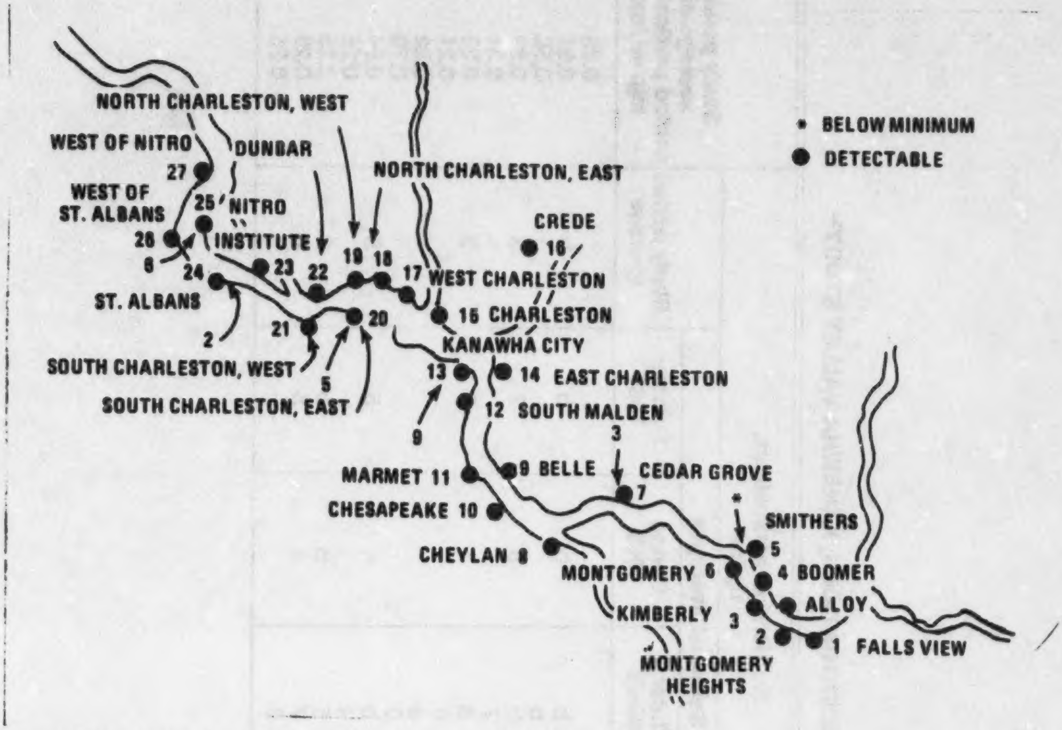
Correlations between vanadium concentrations and meteorological data revealed a strong tendency for vanadium levels to increase with decreasing temperature and increasing atmospheric stability.

5.2.1.2.5. Helsinki — A trace metal investigation sponsored by the World Health Organization was conducted in Helsinki, Finland, during 1962-1963 (Table 5.17).[31] The results of the study prompted the Institute of Occupational Health to investigate vanadium levels in Helsinki. In this second study,[131] monthly dustfall samples were collected at 12 sites within the city from October 1964 to September 1965. Vanadium content of the settled dust was determined by the spectrophotometric technique. The results (Tables 5.18 and 5.19 and Figure 5.6) confirm the expected seasonal pattern in vanadium prevalence. Oil-heating systems are predominant in Helsinki, especially in the center of the city. The vanadium deposition was heaviest in the business district of Aleksanterinkatu, which is consistent with the earlier findings of the World Health Organization.

Table 5.14. VANADIUM CONCENTRATIONS, KANAWHA VALLEY STUDY[24]

Sampling site and number	Suspended particulates, ng/m³					Study period average of settled particulates, mg/m²/mo
	Seasonal averages				Study period average	
	Fall 1964	Winter 1964-65	Spring 1965	Summer 1965		
Falls View (1)						0.35
Smithers (5)	b[a]	b	b	b	b	0.91
Montgomery (6)		b				0.56
Cedar Grove (7)	4	b	b	7	3	0.39
Marmet (11)		7				0.31
Kanawha City (13)	17	6	5	9	9	0.23
Charleston (15)		8				0.21
West Charleston (17)		8				0.32
North Charleston-W (19)		5				0.70
South Charleston-E (20)	11	b	4	5	5	0.94
Dunbar (22)		4				0.26
St. Albans (24)	4	5	b	b	2	0.25
Nitro (25)	10	7	3	5	6	0.28
Nitro-West (27)		8				0.25

[a] b—below minimum detectable level.



* BELOW MINIMUM
● DETECTABLE
NORTH CHARLESTON, WEST
WEST OF NITRO
DUNBAR
NORTH CHARLESTON, EAST
27
25
NITRO
WEST OF ST. ALBANS
26
INSTITUTE
CREDE
16
8
19
18
24
23
22
17
WEST CHARLESTON
ST. ALBANS
21
20
15
CHARLESTON
2
KANAWHA CITY
SOUTH CHARLESTON, WEST
5
13
14
EAST CHARLESTON
SOUTH CHARLESTON, EAST
12
SOUTH MALDEN
9
3
MARMET 11
9 BELLE
CEDAR GROVE
7
CHESAPEAKE 10
SMITHERS
5
CHEYLAN 8
MONTGOMERY 6
4 BOOMER
ALLOY
KIMBERLY
3
2
1
FALLS VIEW
MONTGOMERY HEIGHTS

Figure 5.4. Location of fixed sampling stations in Kanawha River Valley. Average vanadium concentrations (ng/m^3) for the study period (1964-1965) are indicated for selected sites.[27]

Table 5.15. SEASONAL AMBIENT VANADIUM CONCENTRATIONS—BIRMINGHAM, ALABAMA, AREA, 1964-1965[28] (ng/m^3)

Place	Site	Seasonal averages[a]				Study period average
		Spring	Summer	Fall	Winter	
Bessemer	1	5	5	11	b	6
Birmingham	3	4	7	7	b	5
Birmingham		8	7	b	9	7
Birmingham	5	12	7	7	b	7
Birmingham	7	8	3	b	4	4
Fairfield	1	9	4	b	b	5
Irondale	1	4	b	3	b	3
Mt. Brook	1	3	b	3	4	3
Tarrant	1	4	10	8	4	6
Vestavia	1	4	4	3	b	4
Area	—	6	5	5	4	5

[a] b—below minimum detectable level

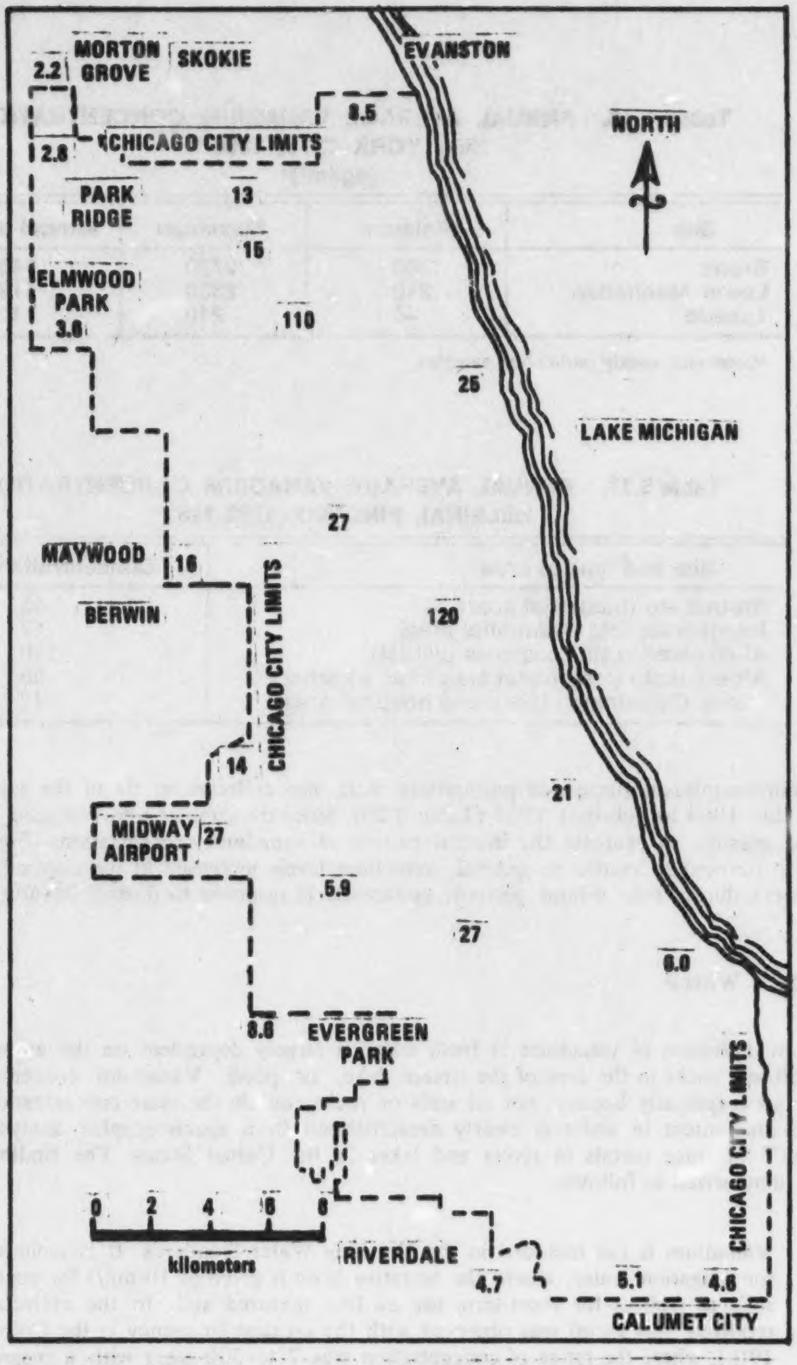

MORTON GROVE
SKOKIE
EVANSTON
2.2
8.5
2.8
CHICAGO CITY LIMITS
NORTH
PARK RIDGE
13
15
ELMWOOD PARK
3.6
110
25
LAKE MICHIGAN
27
MAYWOOD
16
BERWIN
CHICAGO CITY LIMITS
120
14
21
MIDWAY AIRPORT
27
5.9
27
8.0
8.6
EVERGREEN PARK
CHICAGO CITY LIMITS
0
2
4
6
8
kilometers
RIVERDALE
4.7
5.1
4.6
CALUMET CITY

Figure 5.5. Vanadium concentrations for Chicago (24-hour averages in nanograms per cubic meter).[29]

Table 5.16. ANNUAL AVERAGE VANADIUM CONCENTRATIONS, NEW YORK CITY, 1966[30] (ng/m³)[a]

Site	Minimum	Maximum	Annual average
Bronx	600	2790	1460
Lower Manhattan	310	2380	1190
Tuxedo	40	210	120

[a] Continual weekly particulate samples.

Table 5.17. ANNUAL AVERAGE VANADIUM CONCENTRATIONS, HELSINKI, FINLAND, 1962-1963[31]

Site and type of area	Concentration, ng/m³
Sturenkatu (industrial area)	55
Pengerkatu (old residential area)	47
Aleksanterinkatu (business district)	110
Albertinkatu (residential area near a harbor)	68
Minna Canthinkatu (park and hospital area)	42

Six-hour samples of suspended particulates were also collected at six of the sampling sites from December 1964 to February 1965 (Table 5.20). Since the samples were obtained on a 6-hour basis, it was possible to evaluate the diurnal pattern of vanadium concentrations (Figure 5.7). In contrast to particulate matter in general, vanadium levels increased in the evening and early morning hours during four 6-hour periods, apparently in response to diurnal heating patterns.

5.2.2. Water

The concentration of vanadium in fresh water is largely dependent on the amount leached from the soil and rocks in the area of the stream, lake, or pond. Vanadium concentrations in water differ geographically because not all soils or rocks contain the same concentrations. The range of vanadium content in water is clearly demonstrated in a spectrographic analysis by Kopp and Kroner[32] of trace metals in rivers and lakes in the United States. The findings for vanadium were summarized as follows:

> Vanadium is not included in the Drinking Water Standards. It is included in the Criteria for irrigation water, where the tentative limit is given as 10 mg/l for continuous use on all soils as well as for short-term use on fine textured soil. In the collection of data here reported, this metal was observed with the greatest frequency in the Colorado River Basin (9%), where the range of concentration was 7 to 300 μg/l with a mean of 105 μg/l. It was also observed in two samples from the Missouri River Basin at 158 and 184 μg/l. Vanadium has never been detected at measurable concentrations in the Tennessee River Basin, Western Great Lakes Basin, or The Great Basin. It was observed in only 54 of the more than 1,500 samples analyzed for a frequency of 3.4%.

Table 5.18. MONTHLY VANADIUM DEPOSITION, HELSINKI, FINLAND, 1964-1965[31]

Level	Concentration by month and year, mg/m^2-mo											
	10/64	11/64	12/64	8/65	2/65	3/65	4/65	5/65	6/65	7/65	8/65	9/65
Maximum	3.61	7.78	6.96	11.97	9.20	8.11	7.59	6.88	3.10	2.45	3.72	3.17
Minimum	0.49	0.62	0.10	0.19	0.30	0.66	1.11	0.93	0.47	0.44	0.65	0.50
Mean	1.77	2.20	1.63	2.45	2.33	2.64	2.37	2.03	1.31	1.29	1.66	1.61

Table 5.19. VANADIUM DEPOSITION AT VARIOUS INVESTIGATION POINTS, HELSINKI, FINLAND, 1964-1965[31]

Level	Concentration at investigation point, mg/m^2-mo											
	1	2	3	4	5	6	7	8	9	10	11	12
Maximum	2.06	1.28	1.84	1.83	2.63	1.40	4.01	2.43	4.08	4.26	1.97	5.05
Minimum	0.59	0.19	0.33	0.24	0.46	0.10	0.94	0.84	1.25	1.52	2.45	1.47
Mean	1.25	0.57	0.93	1.03	1.29	0.72	2.33	1.64	2.28	2.62	6.19	2.39

Table 5.20. AVERAGE VANADIUM CONCENTRATIONS, DECEMBER TO FEBRUARY 1965, HELSINKI, FINLAND[31]

Investigation point	Number of samples	Vanadium concentrations, ng/m^3		
		Maximum	Minimum	Arithmetic mean
Railway station	15	1,085	57	399
Albertinkatu	12	1,180	3	257
Aleksanterinkatu	13	675	105	259
City Gardens	12	930	022	164
Hameentie	13	700	009	211
Institute of Occupational Health	10	249	7	102

The average for the 54 positive samples in the study was 40 μg/liter, with a range of 2 to 300 μg/liter. The Colorado Plateau is the principal commercial source of vanadium-bearing ores in the United States. The vanadium content of waters draining this area has been studied by Linstedt and Kruger.[33] They employed a more sensitive analytic method (neutron activation) for analysis of their sample than did Kopp and Kroner. Their findings were summarized as follows:

> Vanadium concentrations in samples throughout the Colorado River Basin varied between 0.2 and 49.2 μg/l over the 15-month period of the study. The lowest vanadium concentrations were found in samples from sites on the Animas and Green Rivers. Both these sites are upstream of any major industrial activity. Highest concentration values were found at the sampling sites in the vicinity of the uranium-vanadium milling operations.

Flow levels in the streams and rivers sampled made a noticeable difference in the concentrations observed. The water courses sampled flow into Lake Powell, where they are mixed. Using the data collected and assuming that the vanadium remains in solution once it has entered the fresh water course, the concentration in the lake was calculated to be 3.6 μg/liter. Measurements of the lake effluent showed a level of 3.4 μg/liter, which is in good agreement with measurements from the stream entering the lake.

In fresh waters, vanadium is usually in solution as a salt.[3] Pentavalent vanadium compounds are the most soluble. In going from the rocks and soil into the water, vanadium is oxidized from the trivalent to the pentavalent state. Microorganisms play a significant role in making it more soluble and thus capable of transport.

Durfor and Becker[34] included vanadium in their trace element analyses of drinking water supplies in the United States. Of the samples analyzed, 91 percent were below 10 μg/liter; the maximum level found was 70 μg/liter, and the average was about 4.3 μg/liter.

Much of the soluble vanadium in rivers is removed by precipitation when rivers reach the sea.[3] Bowen[35] estimated that only about 0.001 percent of the vanadium entering the oceans is retained in soluble form in sea water. The total amount of vanadium in the oceans is estimated[3] to be 7.5×10^{12} kg. The concentration of vanadium in sea water ranged from 0.2 to 29 μg/liter. Riley and Taylor[36] made a detailed study of several metallic elements in waters off the African coast south

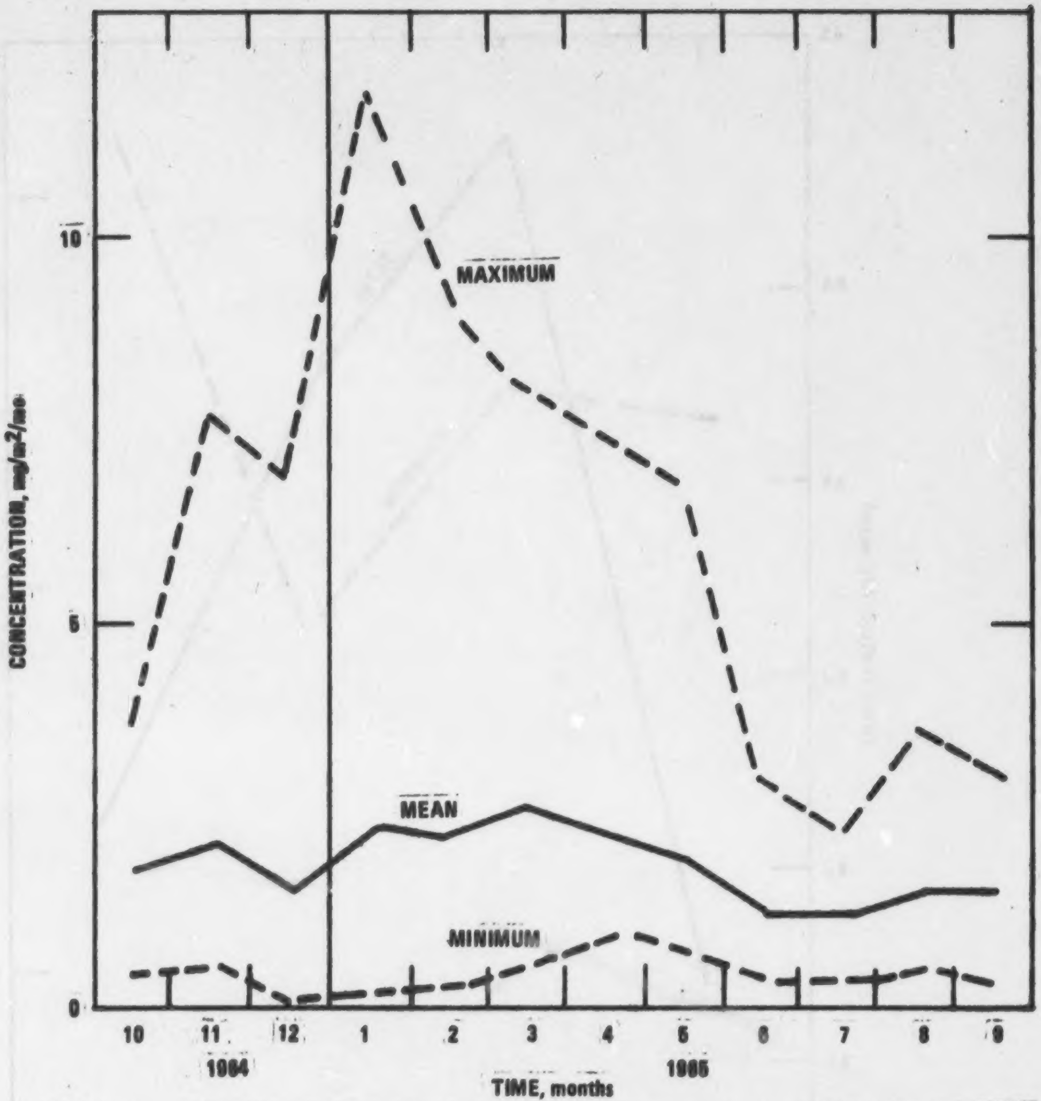


Figure 5.6. Monthly variation of vanadium deposition, Helsinki, Finland, 1964-1965.[31]

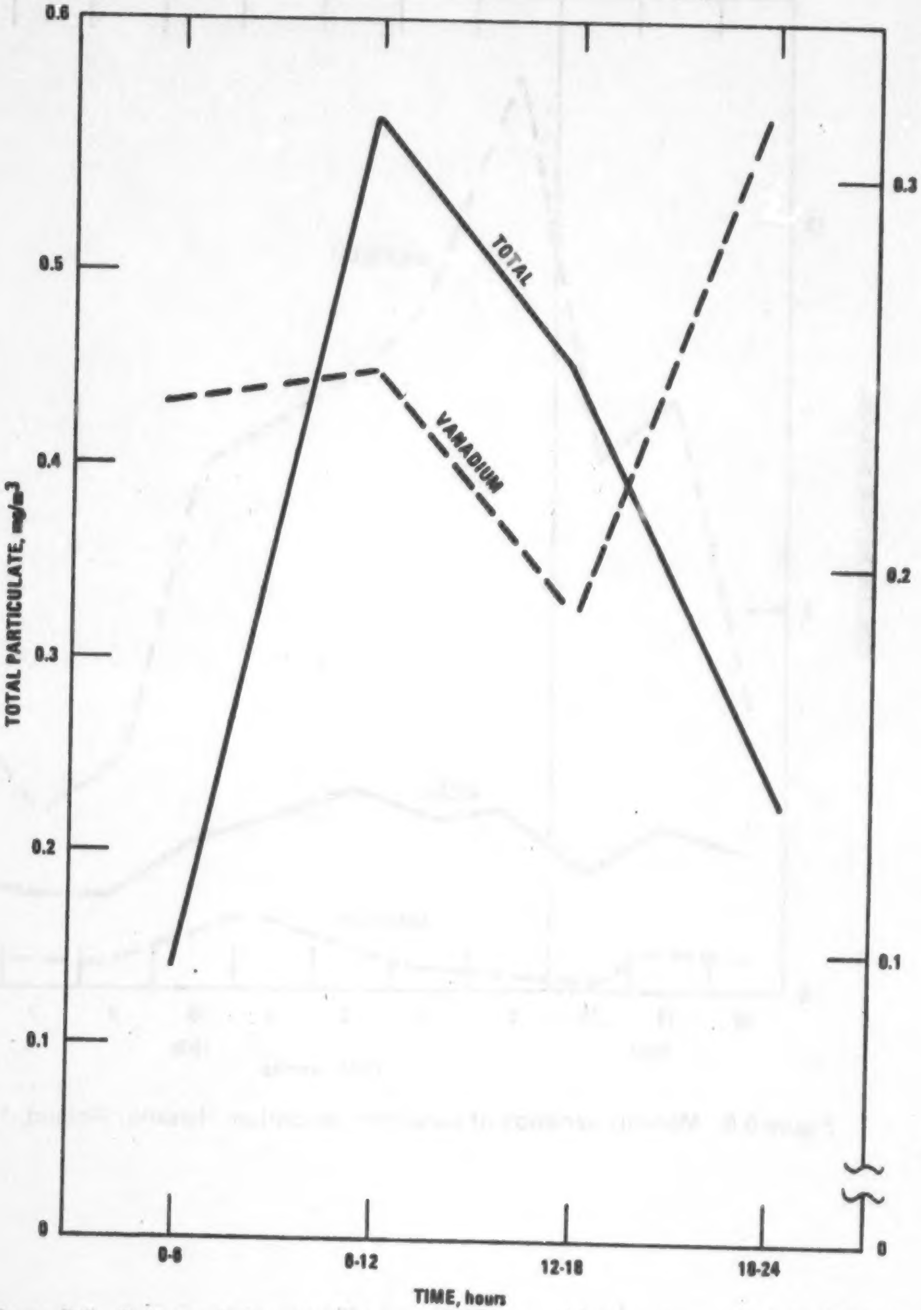


Figure 5.7. Daily variation of total particulate and vanadium concentrations in air of oil-heating area during cold period, Helsinki, Finland, 1964-1965.[31]

of the Canary Islands. Their samples ranged in vanadium content from 0.2 to 5.1 μg/liter, with a mean of 2.1 μg/liter. The standard deviation was large. No pattern of distribution was observed that could be related to the physical variations in the ocean. A biological basis for the patchy distribution was postulated but could not be demonstrated in the study. Krauskopf[37] also postulated a biological basis for the removal of vanadium from ocean water. He found that vanadium was not removed by precipitation as sulfide or other insoluble forms. Also, vanadium was insufficiently removed by adsorption on substances such as manganese or ferric oxides to explain the low levels encountered in the sea. The nature of the postulated organic mechanism of removal of the metal to bottom sediments is not known.

5.2.3. Soil

Information on vanadium in soils, rocks, and sediments may be found in several reviews.[3,38-41] The average concentration of vanadium in the earth's crust is estimated[3] to be 150 mg/kg. The principal source of soil vanadium is the parent rocks from which the soils are formed. Many studies of soil composition have been conducted — so many that it is difficult to compile the data. Almost all soils studied contain small amounts of vanadium; negative findings are thought to result from inadequate methodology. Vanadium levels are reported in the literature to range from 3 to 300 μg/g; concentrations in the United States are at the higher end of the range, those in Japan at the median level, and concentrations in Europe at the lower end of the range. Bertrand[39] indicates that soils originating from Tertiary rocks are lowest in vanadium, Mesozoic-derived soils are somewhat higher, and those of Paleozoic origin are highest.

5.2.4. Food

Ingestion of food and water is the primary source of vanadium in man. For urban populations, the intake of vanadium from air is calculated to be, on the average, only 3 percent. This amount varies, depending on natural concentrations present in water and soil and on degree of industrialization and air pollution. For example, the polluted air of New York City may account for as much as 10 percent of the total intake of vanadium.[42]

Schroeder et al.[43] measured concentrations of vanadium in various categories of foods. Their data indicate that diets containing shellfish, prepared grains, legumes, and leafy vegetables are relatively high in vanadium (Table 5.21). Sardines and herring have been shown to contain 20 μg/100 g when analyzed by colorimetric techniques. Meat and fruit contained little vanadium.

Studies on vanadium content of foodstuffs and its possible role in the etiology of endemic goiter showed this metal to be fairly consistent in two regions sampled.[44] Low levels of 1.46± 0.13 and 0.88 ± 0.7 μg/100 g and high levels of 19.40 ± 3.50 and 22.80 ± 5.75 μg/100 g raw weight were found, respectively, in the two regions sampled. High values were obtained for carrots, cabbage, beans, and sunflowers in both regions.

Fresh milk samples from six locations, taken and analyzed by Söremark,[45] contained about 0.1 ng/g vanadium. Calf liver and pork also had low levels, indicating that many of the most common foods consumed by humans may be rather low in vanadium. The use of emission spectroscopy and

Table 5.21. VANADIUM CONCENTRATIONS IN FOOD[43]

Food category	No. of foods per category	Concentration (wet-weight basis), μg/g	
		Mean	Range
Seafood:			
Shellfish	4	2.39	0.11 to 5.1
Fish	2	0.48	0.0 to 0.96
Meats	8	0.02	0.0 to 0.14
Dairy products	8	0.95	0.0 to 6.52
Grains:			
Whole	14	0.81	0.0 to 2.30
Prepared	6	2.27	0.07 to 6.03
Vegetables:			
Roots	8	0.90	0.0 to 3.02
Legumes	9	2.11	0.0 to 6.00
Leaves	9	1.31	0.55 to 2.53
Fruits	8	0.09	0.0 to 0.36
Nuts	8	0.71	0.0 to 1.96
Beverages	1	0.0	
Condiments	5	0.55	0.06 to 1.36

neutron-activation analysis are improving accuracy and comparability of vanadium data on foods and other biological materials.

Söremark[45] analyzed various food items using neutron-activation analysis (Tables 5.22 and 5.23). Parsley has an unusual affinity for vanadium, 29.5 μg/g, followed by radishes, which averaged 7.9 μg/g in the 10 samples analyzed. The uptake of vanadium-48 from soil by several vegetables also showed parsley and radishes to be high. Meat from North Sea lobsters had the highest level of vanadium (16.1 μg/g) followed by gelatin (2.5 μg/g). All values were based on ash weight.

In the analysis of peas, Mitchell,[46] using emission spectroscopy, found low values for this vegetable, nanograms per gram (wet basis) rather than micrograms per gram as reported by previous investigators. These data, along with neutron-activation analyses by Söremark[45] and Lambert and Simpson[47] tend to support data showing low concentrations in foods.

Unrefined fats and oils, pressed soybeans, corn oil, margarine, pressed linseed, and olive oil showed the highest concentrations of vanadium (Table 5.24.) The refining of fats and oils apparently removes most, if not all, of the vanadium.[43] This was shown in the vanadium level in a sample of fat from a person fed vanadium in soluble form for a period of 18 months. His fat contained three times the amount of vanadium present in a person not fed this diet. Analyses of three refined fats showed no vanadium. Refined egg lecithin and butter also had no detectable vanadium. On the other hand, skim milk had 6.52 μg/g, and egg yolk had 0.68 μg/g on a wet weight basis.[43]

Further evidence of vanadium removal by the refining process was shown with other vegetable oils. All edible unrefined vegetable oils contained vanadium, as did cedar and crude linseed oil. Refined castor oil had no vanadium; and pure vegetable lecithin had barely detectable amounts. Half of the vanadium was lost by refining soybean oil. Extraction of lipid from whole rye flour removed all traces of vanadium.

Table 5.22. VANADIUM CONCENTRATION IN ANIMAL SPECIMENS[45]
($\mu g/g$)

Specimen[a]	Mean, ash-weight basis	Mean, wet-weight basis
Calf liver, Stockholm	0.51	0.01
Calf liver, Boston	0.11	0.0024
Calf flesh, Stockholm	<0.0001	——
Calf teeth, Stockholm	<0.0001	——
Calf bone, Stockholm	<0.0001	——
Pork, Stockholm	<0.0001	——
Fresh trout, soft tissues	0.06	0.0024
Fresh mackerel, soft tissues (North Sea)	0.20	0.0026
Fresh mackerel, bone	2.9	2.0
Sardines, Sweden	0.28	0.0086
Sardines, Norway	0.20	0.0070
Sardines, Portugal	0.46	0.013
Fresh milk, Boston	0.00024	0.000084
Fresh milk, Chicago	0.00016	0.000077
Fresh milk, New York	0.00013	0.000074
Fresh milk, Stockholm	0.00048	0.00011
Fresh milk, Oslo	0.00020	0.00008
Fresh milk, Goeteborg	<0.0001	
Dried skim milk:		
Carnation (U.S.A.)[b]	0.005	0.00023
Starlac[c]	0.00048	0.00019
Famon (Sweden)[d]	<0.0001	
Semper (Sweden)[d]	<0.0001	
Lobster, meat (North Sea)	16.1	0.043
Gelatin (Sweden)	2.5	0.044

[a] Ten samples were taken of each of the specimens listed.
[b] Carnation Food Company, Los Angeles, Calif.
[c] Borden's Food Products, New York, N.Y.
[d] Semper Company, Stockholm, Sweden.

The amounts of vanadium found in foods vary geographically. For example, vanadium has been found in milk in Colorado Springs, Colo., but it was not found in milk from Rochester, New York. Using neutron-activation analysis, vanadium was found at levels of 15 ng/g in random samples of powdered milk taken in San Diego, Calif.[45] Variations were found between milk sampled in Gotenberg, Sweden ($<10^{-4}$ $\mu g/g$), and Boston, Massachusetts (0.24×10^{-3} $\mu g/g$).

For reasons not clear, institutional diets contained 10 times the levels of vanadium normally found in foods.[47] The use of insensitive colorimetric techniques in analyses of these diets may account for this discrepancy and indicates a need to recheck these results.

The amount of vanadium in animal products is primarily from the feed and water ingested, although an unknown amount, probably small, results from the absorption of vanadium from the lung. To accurately assess the contribution that vanadium in food makes to the human body burden of vanadium, comprehensive market basket and drinking water surveys are needed.

Table 5.23. VANADIUM CONCENTRATIONS IN FRUITS AND VEGETABLES[45] ($\mu g/g$)

Specimen[a]	Mean, ash-weight basis	Mean, dry-weight basis	Mean, wet-weight basis
Dill	4.6	0.84	0.14
Lettuce	2.8	0.58	0.021
Parsley[b]	29.5	4.52	0.79
Cucumbers	0.38	0.056	0.0021
Radishes[b]	7.9	1.26	0.0521
Strawberries[b]	0.66	0.031	
Wild strawberries	0.72	0.041	
Red whortleberries	0.54	0.0102	0.0016
Apples[b]	0.33	0.0086	0.0011
Tomatoes[b]	0.041	0.00053	0.000027
Cauliflower	0.093	0.00109	0.000077
Potatoes[b]	0.0093	0.0064	0.00082
Pears	<0.0001	—	—
Carrots[b]	<0.0001	—	—
Common beets	<0.0001	—	—
Peas, frozen	<0.0001	—	—

[a] Ten samples of each item were taken from the Stockholm area.

[b] Five samples of each grown in New Hampshire and five grown in Rhode Island were also analyzed. Vanadium concentrations in these samples were, in general, somewhat lower than those tabulated for the fruits and vegetables grown in Sweden.

Present data on vanadium levels in foods are sparse, and many of the available data may be questioned as to quality. Research is needed to develop reliable techniques for accurately and reproducibly sampling and analyzing levels of vanadium in foods and other biological material and for validating these techniques by actual field surveys.

5.2.5. Plants, Animals, and Man

5.2.5.1. Introduction — Vanadium is found throughout the biosphere. The content in plants, animals, and humans is related to the vanadium in the physical environment on which the biosphere is dependent. Vanadium occurs in a number of valence states and in a variety of materials.[40] It is present in unrefined oils, in vegetable fats, and in many living organisms. Fatty tissues of animals and humans appear to have an affinity for vanadium compounds. Vanadium apparently forms ionic bonds with sulfhydryl groups, which could explain its relatively higher concentrations in hair, keratin, and gelatin.

5.2.5.2. Plants — The concentration of vanadium in water and in soil appear to have the greatest influence on the levels found in plants. Bertrand[39] found vanadium in every sample of the 62 plant species that he analyzed. The mean concentration in higher plants for fresh samples was 0.16 $\mu g/g$ fresh weight, 1.0 $\mu g/g$ dry weight, and 7 $\mu g/g$ in ash. The aerial portions of the plant had the lowest vanadium content and the roots had nearly the same content as the soil in which the plant was growing.

In a more recent study, Hanna and Grant[48] analyzed the foliage of 12 different species of woody ornamental plants to determine their mineral composition. Spectrochemical analyses of 43 different

Table 5.24. VANADIUM CONCENTRATIONS IN FATS AND OILS[43,44]

Fat or oil	Average concentration (wet-weight basis), μg/g
Animal fats	
Mouse	3.06
Rat	0.06
Pig	6.76
Deer	6.82
Beef bone marrow	3.17
Chicken	13.85
Sperm oil	12.67
Human	1.62
Human (fed vanadium for 18 months)	5.13
Lard, refined	0.0
Butter	0.0
Cod liver, refined	0.0
Lecithin, bovine 90 percent	1.28
Egg lecithin, refined	0.0
Mean of fats, raw	6.00
Mean of fats, refined	0.0
Vegetable oils	
Safflower, extracted	2.85
Cottonseed, extracted	3.02
Sunflower	6.95
Vegetable shortening	7.49
Cedar	8.98
Peanut, pressed	10.87
Corn, pressed	11.05–15.80
Linseed, crude, pressed	11.76
Olive, pressed	15.23
Soybean, pressed	43.53
Corn oil margarine	21.75
Mean	14.77
Castor, refined	0.0
Lecithin	
Crude soy	2.45
Refined soy	1.19
Vegetable, pure	0.58
Rye seed flower	
Extracted	5.50
Residue	0.0
Whole	1.24

leaf samples were made for 23 different minerals. The average concentration of vanadium in dry samples was 1.2 μg/g. The authors state that the concentrations of some elements in plant tissue vary with the time of the year, the climate, the soil, the organ sampled, and the position on the plant. Further, they noted wide variations between species as well as within species. Illustrative of this fact was the range of vanadium concentration in pin oak *(Quercus palustris* Muench) — 0.06 to 4.8 μg/g. The lowest and highest levels were found within this species.

Bertrand[39] noted that the root nodules of legumes in which the symbiotic nitrogen-fixing bacterium, *Rhizobium*, is growing contain from 3 to 4 μg/g of vanadium in dry matter.

Vinogradov[38] studied marine organisms to determine a variety of metal concentrations (Table 5.25).

Table 5.25. VANADIUM CONCENTRATIONS IN PLANTS AND ANIMALS[2] (μg/g)

Plant	Average concentration (dry-weight basis)	Animal	Average concentration (dry-weight basis)
Plankton	5	Coelenterates	2.3
Brown algae	2	Annelids	1.2
Bryophytes	2.3	Mollusks	0.7
Ferns	0.13	Echinoderms	1.9
Gymnosperms	0.69	Crustaceans	0.4
Angiosperms	1.6	Insects	0.15
Bacteria	trace	Fish	0.14
Fungi	0.67	Mammals	<0.4

Cowgill[49] analyzed aquatic plants in two freshwater lakes in Connecticut to determine their elemental composition. She found that the vanadium concentration ranged from 0.4 to 80.0 μg/g. Ten specimens of each of six different species were analyzed. The rooted plant species contained the highest vanadium concentrations. Pickerelweed *(Pontedaria cordata* L.) contained the highest concentrations, 80.0 μg/g. None of the other plants tested had levels higher than 5 μg/g. The author suggests that pickerelweed may be an accumulator of vanadium.

Amanita muscaria (L.) Fr., the fly agaric mushroom, is a known accumulator of vanadium. Bertrand[39] noted that it contained approximately 100 times as much vanadium (16 to 181 μg/g dry weight) as other species of fungi.

Mosses have been mentioned as accumulators of vanadium as well as other metals. The concentration of vanadium in mosses, however, is not associated with uptake from the soil, but results from deposition from the air.[50]

5.2.5.3. Animals — Vanadium is found in both terrestrial and aquatic animals (Table 5.26). Bertrand[39] noted that concentrations in vertebrates were so low that it was often difficult to determine if vanadium was present at all.

The concentration of vanadium in sea weeds, mollusks, fishes, and sea water was determined by Fukai and Meinke[51] using neutron-activation analysis. The concentration of vanadium in the soft tissues of fish was determined to be 1,000 times that in sea weed and mollusks.

The highest concentrations of vanadium have been found in the blood of ascidians, certain holothurians, and in the mollusk, *Pleurobranchus plumula*. Concentrations in the ascidians ranged from 3 to 1,900 μg/g, and even 42,000 μg/g has been reported.[52] The holothurian, *Stichopus mobii*, contained 1,235 μg/g, and the mollusk contained 150 μg/g dry weight.[52]

Table 5.26. VANADIUM CONCENTRATIONS IN TISSUES OF WILD ANIMALS[2] (μg/g)

Tissue[a]	Number of samples	Concentration (wet weight basis)	
		Mean	Range
Kidney	4	0.94	0.0-2.07
Liver	4	0.25	0.0-0.94
Heart	4	1.16	0.0-3.40
Spleen	1	1.16	——

[a] Beaver, deer, woodchuck, rabbit, muskrat, and fox.

The fate of vanadium-48-labeled V_2O_5 injected in mice has been studied by Söremark et al.[53] using autoradiographic techniques and scintillation counting at intervals following administration. Skeletal tissues and teeth showed the highest levels. In order of decreasing levels, other bodily tissues and substances containing vanadium were: visceral yolk sac epithelium, lactating mammary glands, renal cortex, liver, lung, skin, salivary glands, intestinal content, muscle, amniotic fluid, heart muscle, feces, cartilage, brain, blood, gastric content, gall bladder content, and spleen.[45] The distribution patterns of radiovanadium were similar to those for mice. The hard and epidermal tissues showed the highest concentrations.[53]

Varying levels of vanadium in drinking water have been administered to hamsters. Relative hepatic ratios remained constant with elapsed time, indicating effective removal by the kidney.[45]

The tissue distribution and percentage retention of vanadium-48 administered intramuscularly in the form of sodium hexavanadate to rats has been studied.[54] The rates were sacrificed 7 days after injection. Percentage of dose retained by various tissues was determined to be: bone, 9.9; liver, 6.2; muscle, 5.0; kidney, 4.4; and blood, 1.5. After a period of 64 days, 10 percent of the injected radiovanadium remained in the rat. Of this amount 4.6 percent was retained by the skeletal tissues.

In acute exposure to carnotite dust, Wilson et al.[55] found lung tissue to retain the highest levels; bone and kidney were found to have small amounts.

Tissue retention of vanadium was determined by ter Heege[56] in cattle poisoned by grass that contained high levels of vanadium. Analysis revealed 24 mg/kg to be present in the liver and 4.3 mg/kg in the renal cortex.

Bertrand[39] reviewed the literature before 1950 pertaining to animal exposure to vanadium. Although much of the early information is conflicting, he considered it likely that vanadium was present in all animals, with the liver and skeletal tissues showing the highest amounts. Although wild animals generally have higher amounts of vanadium in their tissues than humans, very little is found in their lungs. This may be explained by shorter exposure times and also because the air they breathe is less polluted.

The vanadium concentrations of various tissues in wild animals are shown in Table 5.26.

5.2.5.4. ***Humans*** — In humans, vanadium is one of the metals that accumulates in lung tissues with age, an increase that is not evident in other body tissues. It can be inferred, therefore, that vanadium is in the form of insoluble particulates in the lung. Over half of the 78 autopsied lungs analyzed by Tipton and Shafer[57] contained vanadium. Results varied from nondetectable to an extreme of 68 μg/g in dry ash of lung samples. Workers in industry exposed to contaminated dust excreted four times as much vanadium in their urine as did controls. This would indicate that vanadium was absorbed from the lungs and intestines. In an extension of the above study, 168 human lung samples from adults in the United States were analyzed for 24 trace and bulk elements found to increase up to the age of 40.[57]

Some indication that vanadium is absorbed into the blood via the lungs was reported in animal studies by Molfino.[58] He observed that vanadium appeared in the urine shortly after rabbits were exposed to vanadium oxide dust. Massmann[59] also found that 7 days after injecting vanadium pentoxide into rat's trachea, no vanadium remained in the lung. Sjøberg[60] in an inhalation exposure study of rabbits showed only slight retention of the metal in the lungs.

Despite these animal studies showing rapid absorption and little retention of vanadium compounds in the lung, the fact remains that with age, humans do accumulate this metal in lung tissues.[57] Autopsied lung tissues from persons aged 20 to 59 from Middle Eastern countries showed significantly more vanadium than lung tissue taken from persons of similar age who resided in the United States, Africa, and the Far East. Differences in the diets — particularly in unrefined oils, flour, and other foods — could account for these differences.[61]

Concentrations of vanadium and other metals were determined in lung tissues of 65 deceased West Virginia bituminous coal miners.[62] The section of the upper lobe of the left lung closest to the rib cage contained the greatest concentration of trace metals. Hilar lymph nodes of these miners had a higher level of vanadium and free silica than did lung tissues. All trace elements were in greater concentration in the miners than in the same tissues of nonminers, based on values in the literature. Reasonable correlation was shown between air concentrations of vanadium and vanadium content in lungs.

Injection of vanadium-48 in rats also resulted in its accumulation in lung tissues.[53] In the Tipton and Shafer[57] study, paired rank correlation coefficients for concentrations of vanadium and 15 other trace metals with 28 different human body tissues other than lung were not significant. The accumulation of vanadium in the lungs and intestinal tract was similar to that for chromium and manganese, both of which are biologically competitive with vanadium.

The small and large intestines were, next to lung tissues, highest in levels of vanadium[43] and in the frequency of occurrence. Vanadium has an apparent affinity for tissues with high sulfhydryl and triphosphate polar groups. This is indicated by the high levels found in samples of deer hoofs (2.55 μg/g), human hair (2.65 μg/g), and gelatin (12.59 μg/g). According to Schroeder et al.,[43] storage of available vanadium in humans is mainly in fat and serum lipids. Their conclusions were based on these findings: fatty muscle contained 1.2 μg/g but contained none after fat was extracted; human fat contained an average of 1.6 μg/g, whereas fat from a patient receiving vanadium had 5.8 μ/g; and vanadium in serum is removable by chloroform (a fat solvent) but not by trichloroacetic acid or acetone precipitation.

It has been estimated that the standard 70-kg man contains 25 mg of vanadium, with fat containing 22.5 mg and serum, 1.3 mg. The high levels in serum and low levels in urine suggest that either most of the serum is not filtered by the glomeruli or that renal reabsorption is efficient.[43]

Gordos[63] determined levels of vanadium in human hair samples. Depending on the frequency of hair washings, the vanadium content ranged from a few nanograms per gram to 262 ng/g; the mean was 60 ng/g. Older hair samples obtained from private museums dating as early as 1830 showed generally lower values for vanadium. Whether the high levels found in contemporary hair specimens are caused by increased industrial pollution or simply a change in external binding characteristics of the hair specimens after long-term storage is not known. Infrequently washed hair gave higher mean values, approximately 80 ng/g.

The low levels of vanadium detected in human tissues other than bone, skin, and lung indicate a short residence time for the metal.

In the CHESS studies, the amount of vanadium was determined in maternal blood, cord blood, scalp hair, pubic hair, and placenta.[64] These data show low levels in all tissues analyzed. This supports analyses made by other investigators on human tissue specimens from persons not subject to high levels of vanadium exposure. Levels in hair are comparable to those found by Gordos.[63]

A number of studies [65-67] have been directed toward determining the effectiveness of vanadium compounds in reducing cavities in animal and human teeth. Analyses of teeth showed traces of vanadium, with the largest quantity shown in molars.

Table 5.27 presents a summary of vanadium levels determined by Tipton and Cook[68] in human tissue specimens.

5.3 TRANSPORT AND MODELING

5.3.1. Transport

An accurate description of the transformation and transport of pollutants emitted into the environment is needed so that the quality of the environment can be related to the sources of pollution and so that predictions can be made on the emission control required under present and future conditions. When particles are present, those processes that affect the size distribution spectrum in the air parcel must be described. Processes affecting the aerosol spectrum near the earth's surface include:[69]

- Production of aerosols at the surface by natural and artificial sources.
- Growth of particles by heterogeneous gas reactions on the surface of particles.
- Production and growth of particles by homogeneous gas reactions and subsequent attachment of reaction products to the aerosols.
- Gain or loss of particles entering by diffusion or convection from neighboring air.
- Net change in particle concentrations by thermal (Brownian) coagulation.
- Net change of particle concentrations by collisions between particles resulting from turbulent velocity gradients.

- **Loss of particles by gravitational sedimentation.**
- **Loss of particles by impaction on obstacles at the earth's surface.**
- **Loss of particles by washout under clouds.**
- **Loss of particles by rainout inside clouds.**

Table 5.27. VANADIUM CONCENTRATIONS IN HUMAN TISSUES[68]

Tissue	Samples positive for V/ samples analyzed	Concentration (ash weight), $\mu g/g$ Median	Maximum
Adrenal	1/13	<1	<1
Esophagus	5/66	<1	<1
Stomach	3/130	<1	<1
Duodenum	5/67	<1	<1
Jejunum	4/102	<1	<1
Ileum	31/84	<1	3
Cecum	14/31	<1	4
Sigmoid colon	17/108	<1	3
Rectum	11/42	<1	2
Kidney	0/144	—	—
Liver	5/148	<1	<1
Heart	0/143	—	—
Lung	73/141	<1	13
Omentum	11/75	<1	3
Pancreas	2/139	<1	<1
Prostate	1/50	<1	<1
Spleen	1/143	<1	<1
Skin	3/21	<1	<1
Thyroid	1/21	<1	<1
Bladder	3/110	<1	<1
Uterus	1/32	<1	<1

Hidy[70] has considered those processes that contribute directly to the aging (change *in situ* of physical or chemical properties) of aerosols in the atmosphere and has attempted to estimate the relative importance of these mechanisms for three different particle size classes. From existing data, he was unable to estimate the importance of heterogeneous gas-surface reactions and the formation of particles by homogeneous gas reactions. His calculations demonstrate that thermal coagulation and turbulent diffusion dominate the aging mechanism for particles near the ground. Gravitational sedimentation is relatively unimportant for particles smaller than about 10 μm radius but becomes increasingly more significant as the particle radius increases.

Little information is available on the detailed mechanism by which airborne vanadium particles participate in these processes. Furthermore, the ultimate fate of vanadium in the atmosphere is not known. Vanadium residence time in the lower Delaware River Valley air shed, however, has been roughly estimated to be 0.1 and 1 day.[6]

5.3.2. General Particulate Modeling

Modeling efforts for particulates, including vanadium-containing particles, must consider the following transport, transformation, and removal processes.

5.3.2.1. Sedimentation — Gravitational sedimentation, although relatively unimportant for particles less than 10 μm radius, becomes increasingly more important as the particle size increases above 10 μm. Large vanadium particles emitted into the ambient air would be expected to fall out in the near vicinity of their source.

5.3.2.2. Diffusion and Transport Processes — The transport of aerosol particles from one place to another is controlled primarily by prevailing winds. Diffusion by Brownian motion is insignificant when compared with the convection diffusion produced by turbulent air motion. Rural areas on the eastern coast from Maine to South Carolina show a significantly higher concentration of vanadium than other rural areas of the country. This finding suggests that vanadium emitted in urban areas from the combustion of oil and coal is being transported to nonurban areas.

Little information is available on the transport of vanadium by groundwater. Although vanadium pentoxide is not highly soluble in water (0.8 g/100 cm^3 water at 20°C), its solubility would probably increase significantly in an acid rain or if the oxide were converted to a sulfate such as vanadyl sulfate. Microbial action is believed to assist in the conversion of vanadium into more soluble forms for transport in groundwater.[1]

5.3.2.3. Coagulation Processes — Coagulation processes are known to affect aerosol distribution. Hidy's calculation[70] suggests that thermal coagulation is of primary importance for the smallest particles near the ground. For particles larger than 0.5 μm radius, thermal coagulation decreases in importance as the particle size increases. Turbulent coagulation is comparatively weak for particles less than 0.5 μm, but it becomes more significant for larger particles. However, near the ground, where particle concentrations are high and turbulence intensity is great, turbulent coagulation may provide an effective mechanism for growth between the range where thermal coagulation dominates to the range where sedimentation dominates.

5.3.2.4. Heterogenous Gas Reactions on Surfaces of Particles — Particles in the atmosphere are thought to be involved in many chemical reactions associated with photochemical smog. The oxidation of gases such as sulfur dioxide is accelerated in the presence of particles; and the formation of nitric acid, by the reaction of nitrogen pentoxide with water, is believed to be a surface reaction. Although vanadium is frequently used as a catalyst in industrial applications, its catalytic reactivity in the ambient air is unknown.

5.3.2.5. Scavenging — The scavenging of small particles by large particles falling through an air parcel is thought to be negligible.

5.3.2.6. Washout and Rainout — Although the precipitation removal processes operate only intermittently, they represent an important factor. Washout has been defined as the removal of particles by sweepout by precipitation under clouds; whereas rainout refers to removal processes taking place within clouds. Hidy's[70] calculations suggest that washout may be less important than other removal mechanisms near the ground, but that it becomes more significant at altitudes above 1 km.

5.3.2.7. Impaction and Diffusional Deposition on Surfaces — Particles will be deposited on a surface as a result of Brownian diffusion towards the surface and as a result of impaction. The larger, heavier particles may be deposited on the surface; but smaller, lighter particles may not reach the surface because they are dragged around by the deflected air before they contact the surface.

5.3.2.8. Reentrainment — The possibility of reentrainment of particles located on surfaces must be considered. The importance of this mechanism for reentrainment of vanadium from dust and soils has not been assessed.

5.3.2.9. Microbial Action — Concentrations of vanadium in water and soil appear to have a large influence on microbial action. Specific auxotrophic bacteria oxidize the reduced forms of vanadium to obtain a part or all of their energy for growth and multiplication.[1] Goren[71] has coupled the iron-oxidizing bacteria with the oxidation of vanadium in acidic leaching solutions. The biogeochemical vanadium cycle in Figure 5.8 denotes these interrelationships.[1] *Thiobacillus ferrooxidans* and *Ferrobacillus thiooxidans* catalyze the oxidation of iron, which in turn oxidizes vanadium. The sulfate-reducing bacteria generate hydrogen sulfide and provide conditions for the reduction of pentavalent vanadium and the formation of vanadium sulfides. *Micrococcus lactilyticus* catalyzes the reduction of vanadium sulfide, and trivalent vanadium is formed. The mineral minasragrite is probably formed by the microbial oxidation of vanadium sulfide.

5.3.3. Vanadium Model

Although development of a detailed model describing the mechanisms by which airborne vanadium particles precipitate in the above process is beyond present capabilities, Tullar and Suffet[6] have put forth an elementary model of the fate of vanadium in an urban air shed (Figure 5.9). The shed covers an area approximately 40 km long, from Philadelphia to Delaware City, reaching 12 km on either side of the river for a 24-km width. An arithmetic mean vanadium concentration of 0.23 $\mu g/m^3$ is used for the model area for 1967.

The model assumed that the only significant emissions come from combustion of vanadium-rich fuel oils in refineries, power plants, and other industrial sources. Approximately 3.8×10^6 Mg/yr (MT/yr) of vanadium- rich oil were burned. Using an estimate of 1 Mg (MT) of particulates for each 1,000 Mg (MT) of fuel oil and 5 percent vanadium content in the fly ash, they calculated 190 Mg (MT) of vanadium in the 1967 fly ash emissions in the model area. Approximately 4×10^6 Mg (MT) of vanadium-rich crude were processed by fluid catalytic cracking units in the area during 1967. Using an estimate of 190×10^{-6} Mg (MT) of particulate emisions per Mg (MT) of crude oil processed, and assuming a 5-percent vanadium content in the fly ash, 43 Mg (MT) were added to the emission inventory. The total estimated vanadium emission from the model area was then estimated to be 233 Mg (MT) for the year.

The size breakdown for the total emissions was estimated (Table 5.28). These results do not agree with the 1970 NASN study (Table 5.13), which showed that approximately 80 percent of the suspended vanadium particulate had a MMD of less than 2 μm.

Consideration of the chemistry occurring within power plants indicated that the dominant vanadium compound in industrial emissions was particulate V_2O_5.

An order of magnitude calculation of the residence time within the first kilometer of the atmosphere was made. Using a mean vanadium concentration of 0.23 $\mu g/m^3$, the mass of vanadium in the 1,000-km^2 air shed was 0.23 Mg (MT). Dividing this value by a total emission of 233 Mg/yr (MT/yr) gives a residence time of 0.36 day. Because of the approximate nature of the calculation, it was concluded that the residence time was less than 1 day.

Based on these considerations, Tullar and Suffet[6] have developed a diagram depicting the fate of vanadium in an urban air shed (Figure 5.9).

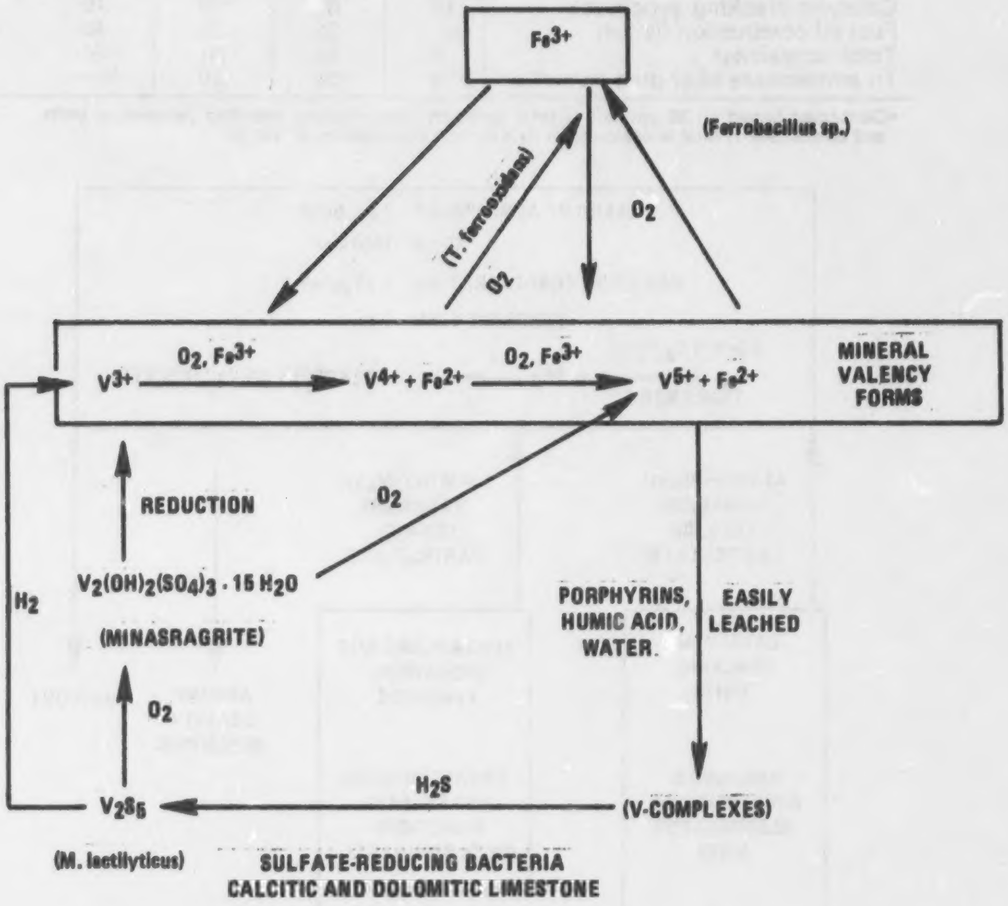

Fe^{3+}
(Ferrobacillus sp.)
(T. ferrooxidans)
O_2
O_2
O_2, Fe^{3+}
V^{3+}
$V^{4+} + Fe^{2+}$
O_2, Fe^{3+}
$V^{5+} + Fe^{2+}$
MINERAL VALENCY FORMS
REDUCTION
O_2
H_2
$V_2(OH)_2(SO_4)_3 \cdot 15\,H_2O$
(MINASRAGRITE)
PORPHYRINS, HUMIC ACID, WATER.
EASILY LEACHED
O_2
H_2S
V_2S_5
(V-COMPLEXES)
(M. lactilyticus)
SULFATE-REDUCING BACTERIA
CALCITIC AND DOLOMITIC LIMESTONE

Figure 5.8. Biogeochemical vanadium cycle.[6]

Table 5.28. ESTIMATED PARTICLE SIZE DISTRIBUTION[a]
(weight percent)

Emissions	Particle size range, μm			
	<2	2-5	5-10	10-20
Catalytic cracking processes	12	65	13	10
Fuel oil combustion fly ash	—	25	35	40
Total emissions[a]	2	33	31	34
To atmosphere after dust collection	20	60	20	—

[a] Calculated based on 20 percent of total emission from catalytic cracking processing units and 80 percent of total emission from fly ash from combustion of fuel oil.

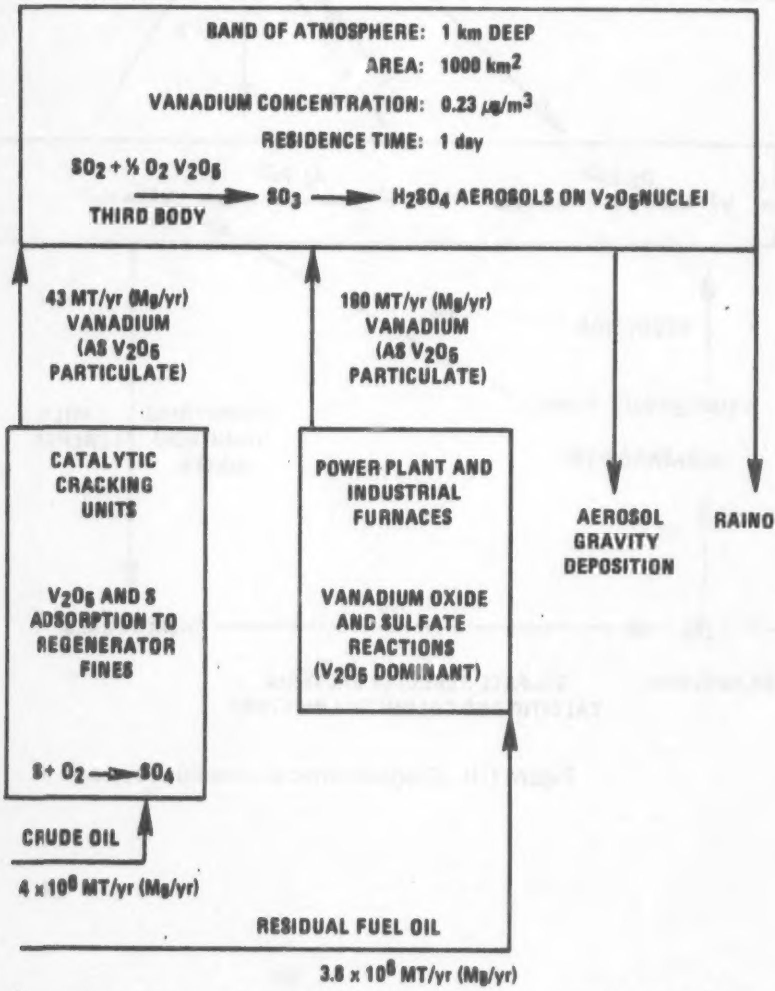

BAND OF ATMOSPHERE: 1 km DEEP
AREA: 1000 km²
VANADIUM CONCENTRATION: 0.23 µg/m³
RESIDENCE TIME: 1 day
SO₂ + ½ O₂ V₂O₅
THIRD BODY
SO₃
H₂SO₄ AEROSOLS ON V₂O₅NUCLEI
43 MT/yr (Mg/yr) VANADIUM (AS V₂O₅ PARTICULATE)
160 MT/yr (Mg/yr) VANADIUM (AS V₂O₅ PARTICULATE)
CATALYTIC CRACKING UNITS
V₂O₅ AND S ADSORPTION TO REGENERATOR FINES
S + O₂ → SO₄
POWER PLANT AND INDUSTRIAL FURNACES
VANADIUM OXIDE AND SULFATE REACTIONS (V₂O₅ DOMINANT)
AEROSOL GRAVITY DEPOSITION
RAINOUT
CRUDE OIL
4 x 10⁶ MT/yr (Mg/yr)
RESIDUAL FUEL OIL
3.8 x 10⁶ MT/yr (Mg/yr)

Figure 5.9. Flow diagram of fate of vanadium in urban air shed.[6]

5.4 REFERENCES FOR SECTION 5

1. Zajic, J. E. Microbial Biogeochemistry. New York, Academic Press, 1969. 345 p.

2. Schroeder, H. A. Air Quality Monograph, Monograph No. 70-13, Vanadium. American Petroleum Institute, Washington, D. C., 1970. 32 p.

3. Vanadium. National Academy of Sciences, Committee on Biologic Effects of Atmospheric Pollutants. Washington, D. C., June 1973.

4. National Inventory of Sources and Emissions: Arsenic, Beryllium, Manganese, Mercury and Vanadium; V. Vanadium. W. E. Davis and Associates, Leawood, Kansas, June 1971.

5. Bowden, A. T., P. Draper, and H. Rassling. The Problem of Fuel Oil Deposition in Open-Cycle Gas Turbines. Proc. (A), Inst. Mech. Eng. *167*:291-312, 1953.

6. Tullar, I. V., and I. H. Suffet. The Fate of Vanadium in an Urban Air Shed, the Lower Delaware River Valley. (Presented at the 66th Annual Meeting of the Air Pollution Control Association. Chicago, Ill., June 24, 1973.) Paper 73-117.

7. Gerstle, R. W., S. T. Cuffe, A. A. Orning, and C. H. Schwartz. Air Pollutant Emissions from Coal-Fired Power Plants, Report No. 2. J. Air Pollut. Contr. Ass. *15*(2):59-64, 1965.

8. Radford, H. D., and R. C. Rigg. New Way to Desulfurize Residuals. Hydrocarbon Process. *49*:187-191, 1970.

9. Lee, R. E., and D. J. von Lehmden. Trace Metal Pollution in the Environment. J. Air Pollut. Contr. Ass. *23*:853, October 1973.

10. Bolton, N. E., W. L. Lyon, R. I. Van Hook, A. W. Andren, W. Fulkerson, J. A. Carter, and J. F. Emery. Trace Element Measurements at the Coal-Fired Allen Steam Plant, Progress Report June 1971-January 1973. Oak Ridge National Laboratory, Oak Ridge, Tennessee, ORNL-NSF-EP-43, March 1973. 83 p.

11. Von Lehmden, D. J. Unpublished data, U. S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N. C., August 1973.

12. Jungers, R. H. Unpublished data. U. S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N. C., 1973.

13. Bryan, D. E., V. P. Gwinn, and H. R. Lukens. Development of Nuclear Analytical Techniques for Oil Slick Identification. Phase 1. Gulf General Atomic. Report 9889, AEC Contract AT(04-3)-167, San Diego, January 1970.

14. Van Dyke, L. F. U. S. Power Firms Begin Burning Crude. Oil and Gas. J. *70* (6):28-30, February, 1972.

15. Ruch, R. R., H. J. Gluskoter, and N. F. Shimp. Occurrence and Distribution of Potentially Volatile Trace Elements in Coal-Interim Report, Illinois State Geological Survey. Environmental Geology Notes. No. 61, April 1973.

16. Jungers, R. H., R. E. Lee, and D. J. von Lehmden. The EPA National Fuels Surveillance Network. I. Trace Constituents in Gasoline and Gasoline Fuel Additives. Environ. Sci. Health Perspectives Technol. (In Press).

17. Jungers, R. H. Analysis of Vanadium in Fuels. U. S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N. C. Unpublished, 1973.

18. Whitman, N. E. Unpublished data provided to the ERC Task Force on Vanadium. Bethlehem Steel Corporation, Bethlehem, Pa., 1973.

19. Anderson, D. M. Unpublished data provided to the ERC Task Force on Vanadium. Bethlehem Steel Corporation, Bethlehem, Pa., 1973.

20. DeHuff, G. L. Vanadium. *In:* Minerals Yearbook, 1968. Department of Interior, Bureau of Mines, Washington, D. C., 1969. p. 1143.

21. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment With Intended Changes for 1972. American Conference of Governmental Industrial Hygienists, Cincinnati, 1972.

22. Hoffman, G. L., R. A. Duce, and W. H. Zoller. Vanadium, Copper, and Aluminum in the Lower Atmosphere between California and Hawaii. Environ. Sci. Technol. *3*:1207-1210, November 1969.

23. Rhan, K. A. Sources of Trace Elements in Aerosols: An Approach to Clean Air. Department of Meteorology, University of Michigan, Ann Arbor, Mich. U. S. Atomic Energy Commission Contract No. COO-1705-9, 1971. 309 p.

24. NASN Vanadium Data Stored in the National Aerometric Data Bank. Unpublished data. U. S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N. C. 1973.

25. Athanassiadis, Y. C. Preliminary Air Pollution Survey of Vanadium and Its Compounds; A Literature Review. U. S. Department of Health, Education, Welfare, Public Health Service, National Air Pollution Control Administration, Raleigh, N. C. NAPCA Publication No. APTD 69-48, October 1969. 91 p.

26. Lee, R. E., S. S. Goranson, R. E. Enrione, and G. B. Morgan. National Air Surveillance Cascade Impactor Network. II. Size Distribution Measurements of Trace Metal Components. Environ. Sci. Technol. *6*:1025-1030, 1972.

27. Kanawha Valley Air Pollution Study. U. S. Department of Health, Education, and Welfare. Public Health Service. National Air Pollution Control Administration, Raleigh, N. C. NAPCA Publication No. APTD 70-1, March 1970.

28. Hauser, T. R., J. J. Henderson, and F. B. Benson. The Polynuclear Hydrocarbon and Metal Concentration of the Air over the Greater Birmingham Area. In-house report. U. S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N. C. April, 1971.

29. Brar, S. S., D. M. Nelson, E. L. Kanabrocki, C. E. Moore, C. D. Burham, and D. M. Holton. Thermal Neutron Activation Analysis of Particulate Matter in the Surface Air of the Chicago Metropolitan Area. Environ. Sci. Technol. *4*:5-54, 1970.

30. Kneip, T. J., M. Eisenbud, C. D. Strehlow, and P. C. Freundenthal. Airborne Particulates in New York City. J. Air Pollut. Contr. Ass. *20*(3):144-149, 1970.

31. Laamanen, A., A. Lofgren, and L. Noro. Vanadium: A Specific Pollutant in Air of Helsinki. Institute of Occupational Health. Helsinki, Finland, Report No. 40, March, 1966.

32. Kopp, J. F., and R. C. Kroner. Trace Metals in Water of the United States. Federal Water Pollution Control Administration, Cincinnati, Ohio. 1968.

33. Linstedt, K., and P. Kruger. Vanadium Concentrations in Colorado River Basin Waters. Amer. Water Works Ass. J. *61*(2):85-88, 1969.

34. Durfor, C. N., and E. Becker. Public Water Supplies of the 100 Largest Cities in the U. S., 1962. Geological Survey, Washington, D. C. Water Supply Paper No. 1812, 1963.

35. Bowen, H. J. M. Trace Elements in Biochemistry. London, Academic Press, 1966. 241 p.

36. Riley, J. P., and D. Taylor. The Concentrations of Cadmium, Copper, Iron, Manganese, Molybdenum, Nickel, Vanadium, and Zinc in Part of the Tropical North-East Atlantic Ocean. Deep Sea Res. *19*:307-317, 1972.

37. Krauskopf, K. B. Factors Controlling the Concentration of Thirteen Rare Metals in Sea Water. Geochim. Cosmochim. Acta. *9*:1-32B, 1956.

38. Vinogradov, A. P. The Geochemistry of Rare and Dispersed Chemical Elements in Soils, 2nd Ed. Translated from Russian by Consultants Bureau, Inc., New York. 1959. 209 p.

39. Bertrand, D. Survey of Contemporary Knowledge of Biogeochemistry. 2. The Geochemistry of Vanadium. Bull. Amer. Museum of Natural History. *94*:403-455, 1950.

40. Cannon, H. L. The Biogeochemistry of Vanadium. Soil Sci. *96*:196-204, 1963.

41. Pratt, P. F. Vanadium. *In:* Diagnostic Criteria for Plants and Soils. Chapman, H. D. (ed.), Riverside, Calif.; University of California Press, 1966.

42. Tipton, I. H., P. L. Stewart, and J. Dickson. Patterns of Elemental Excretion in Long-Term Balance Studies. Health Phys. *16*:455, 1969.

43. Schroeder, H. A., J. Balassa, and I. H. Tipton. Abnormal Trace Metals in Man: Vanadium. J. Chron. Dis. *16*:1407, 1963.

44. Barannik, P. I. et al. Levels of Trace Elements and Natural Radioactivity of Food Products of Some Areas of the Kiev Region. (Translated from Russian). Vop. Pitan. (Moscow) *29*(1):79-81, 1970.

45. Soremark, R. Vanadium in Some Biological Specimens. J. Nutr. *92*:183-190, 1967.

46. Mitchell, P. L. Emission Spectrochemical Analysis. *In:* Determination of Trace Elements in Plants and Other Biological Material. New York, John Wiley and Sons, Inc., 1957. p. 398-412.

47. Lambert, J. P. F., and R. E. Simpson. Determination of Vanadium by Neutron Activation Analysis at Nanogram Levels to Formulate a Low-Vanadium Diet. J. Ass. Off. Anal. Chem., *53*:1145-1150, 1970.

48. Hanna, W. J., and C. L. Grant. Spectrochemical Analyses of Certain Trees and Ornamentals for 23 Elements. Bull. Torrey Bot. Club. *89*:293-302, 1962.

49. Cowgill, U. M. The Determination of All Detectable Elements in the Aquatic Plants of Linsley Pond and Cedar Lake (N. Bradford, Conn.) by X-Ray Emission and Optical Emission Spectroscopy. Appl. Spectrosc. *27*: 5-9, 1973.

50. Tyler, G. Heavy Metals Pollute Nature, May Reduce Productivity. Ambio. *1*:52-59, 1972.

51. Fukai, R., and Meinke, W. W. Activation Analyses of Vanadium, Arsenic, Molybdenum, Tungsten, Rhenium and Gold in Marine Organisms. Limnol. Oceanogr. *7*:186, 1962.

52. Underwood, E. J. Trace Elements in Human and Animal Nutrition, 3rd Ed. New York, Academic Press, 1971.

53. Soremark, R., S. Ullberg, and L. E. Applegren. Autographic Localization of V-48 Labelled Vanadium Pentoxide in Developing Teeth and Bones of Rats. Acta Odont. Scand. *20*:225-232, 1963.

54. Strain, W. H., W. P. Berliner, C. A. Lankav, R. K. McEroy, W. J. Pories, and R. H. Greenlaw. Retention of Radioisotopes by Hair, Bone and Vascular Tissue. J. Nucl. Med. *5*:664674, 1964.

55. Wilson, H. B., H. E. Stokinger, and G. E. Sylvester. Acute Toxicity of Carnotite Ore Dust. Arch. Ind. Hyg. 7:301, 1963.

56. ter Heege, J. H. Een Intoxicate bij Runderen Door Opname van Stookolieroet (Poisoning of Cattle by Ingestion of Fuel Oil) (Transl. from Dutch). Soot. Tijdschr. Diergenees (Rotterdam) *89*(18):1300-1304, 1964.

57. Tipton, I. H. and J. J. Shafer. Statistical Analysis of Lung Trace Element Levels. Arch. Environ. Health. *8*:58-67, 1964.

58. Molfino, F. Contributi Sperimentale Allo Studio dell'Intossicazione Professionale da Vanadio (Experiments on Occupational Vanadium Poisoning). Rass. Med. Industr. (Turin) *9*:362, 1938. English abstract in J. Ind. Hyg. *21*:96, 1939.

59. Massmann, W. Experimental Untersuchungen uber die biologische Wirkung von Vanadinverbindungen (Experimental Investigations on the Biological Activity of Vanadium). Arch. Toxikol. (Berlin) *16*:182, 1956.

60. Sjoberg, S. G. Vanadium Pentoxide Dust; A Clinical Experimental Investigation on Its Effects after Inhalation. Acta. Med. Scand. (Suppl. 238) *138*:1-188, 1950.

61. Tipton, I. H., H. A. Schroeder, H. M. Perry, Jr., and M. J. Cook. Trace Elements in Human Tissue. Part III. Subjects from Africa, the Near and Far East and Europe. Health Phys. *11*:403, 1965.

62. Carlberg. J. R., J. V. Crable, L. P. Limtiaca, Harold B. Norris, John L. Holtz, and F. R. Woolwicz. Total Dust, Coal, Free Silica and Trace Metal Concentrations in Bituminous Coal Miners Lungs. Amer. Ind. Hyg. Ass. J., *32*:432-440, July 1971.

63. Gordos, A. A. University of Michigan, Ann Arbor, Mich. Verbal communication on results of hair as an indicator of trace metal body burden, with D. Worf, Human Studies Laboratory, U. S. Environmental Protection Agency, Research Triangle Park, N. C., June 1973.

64. Trace Metal Analysis of Maternal-Fetal Tissue Sets, Human Scalp Hair, and Housedust. Stewart Labs., Inc. Knoxville, Tenn. Work performed under EPA Contract No. 68-02-0663, December, 1972.

65. Grippaudo, G. et al. Studies on Vanadium and Experimental Caries. Annal. di Stomatologia. (Rome) *15*:87-94, 1966.

66. Lowater, F. Chemical Composition of Teeth. Biochem. J. *31*:837, 1937.

67. Thomassen, P. R., and H. M. Leicester. Uptake of Radioactive Beryllium, Vanadium, Selenium, Cerium, Yttrium in the Tissues and Teeth of Rats. J. Dent. Res. *43*:346, 1964.

68. Tipton, I. H., and M. J. Cook. Trace Elements in Human Tissues. II. Adult Subjects from the U. S. Health Phys. *9*:103, 1963.

69. Junge, C. Comments on "Concentrations and Size Distribution Measurements of Atmospheric Aerosols and a Test of the Theory of Self Preserving Size Distribution." J. Atmos. Sci. *26*:603-608, May 1969.

70. Hidy, G. M. The Dynamics of Aerosols in the Lower Troposphere. *In:* Assessment of Airborne Particles. Mercer, T. T. (ed.). Springfield, Ill., C. C. Thomas, 1972. p. 81-115.

71. Goren, M. Oxidation and Recovery of Vanadium Values from Acidic Aqueous Media. U. S. Patent 3, 252, 756. 1966.

6. EFFECTS OF VANADIUM

6.1 BIOMEDICAL EFFECTS

6.1.1. General Introduction

Studies in animals and man have shown that the toxicity of vanadium compounds is principally a function of the route of administration or exposure. Vanadium salts are considered highly toxic in a number of species when given by intravenous or subcutaneous injection. Inhalation of vanadium compounds produces moderate toxicity with marked irritation to the respiratory tract.[1] Since most vanadium compounds are poorly absorbed from the gastrointestinal tract, they exhibit a low order of toxicity by the oral route.[2] Reviews on the toxic and irritative properties of vanadium compounds have been published by Symanski,[3] Sjøberg,[4] Hudson,[1] Stokinger,[5] Athanassiadis,[6] and Schroeder.[7] The following is a limited survey of the more pertinent literature on routes of exposure to, metabolism of, and health effects of vanadium in man and experimental animals.

6.1.2. Human Exposure

6.1.2.1. Industrial Exposure — Vanadium and its compounds possess many valuable chemical and physical properties that have resulted in increased production and use in recent years. From the point of view of industrial hygiene, the most important vanadium compounds are vanadium pentoxide, vanadium trioxide, ferrovanadium, vanadium carbide, and vanadium salts, such as sodium and ammonium vanadate. The oxides and salts are commonly used in industry in the powder form, which entails the possibility of dust and aerosol formation when the substances are crushed or ground. Many metallurgical processes involve production of vapors containing vanadium pentoxide, which condense to form respirable aerosols. Boiler-cleaning operations generate dusts containing the pentoxide and trioxide compounds. Combustion of residual fuels having a high vanadium content is likely to produce aerosols of the pentoxide as well as oxide complexes of vanadium with other metals. (Details on concentration and duration of industrial exposure are given in Section 6.1.4.)

The portal of entry for vanadium in most industrial situations is the respiratory tract. The gastrointestinal tract is also an important portal of entry in the case of dusts and larger particles that are coughed up and swallowed. Soluble forms of vanadium can penetrate the skin,[5] although this route of exposure appears to be of smaller significance industrially.

6.1.2.2. Nonindustrial Exposure — The major route of nonindustrial exposure to vanadium is via the gastrointestinal tract from food and water. Tipton et al.[8] determined the intake for 347 days of vanadium from food and water by two men aged 23 and 25 (Table 6.1). The calculated intake of vanadium from water is 4 percent of that from food; whereas the intake from air is about 3 percent of that from food. The proportion of vanadium supplied in water varies from 0 to about 7 percent, depending on the water supply. Similarly, the proportion of the total vanadium intake attributable to inhaled air varies from zero in most parts of the country to around 10 percent in New York City.

Table 6.1. BALANCE OF VANADIUM IN TWO MEN[7,8]

Item	Concentration of vanadium, µg/day (mean)	
	Subject C[a]	Subject D
Input:		
Diet[b]	61	170
Air[c]	2	5
Total	63	175
Output:		
Feces	120	150
Urine	18	23
Total	138	173
Balance	-75	2

[a] Contribution of vanadium from seasoning in diet may have been omitted during first three-fourths of the study.
[b] About 4 percent of vanadium in diet is from drinking water.
[c] Intake from air is about 3 percent of that from food.

According to Schroeder,[9] the average daily intake of vanadium from food and water is 116 µg. On the basis of 1966 NASN data, Schroeder[9] calculated (Table 6.2) that the maximum intake of vanadium from air in 58 cities of the United States and Puerto Rico is 9.16 µg. Thus, in this

Table 6.2. VANADIUM INTAKE IN RESIDENTS OF URBAN AND NONURBAN AREAS[9,a]

Item	Amount
Cities studied	44
Nonurban areas studied	29
Range, urban, µg/m^3	0.001 to 0.458
Range, nonurban, µg/m^3	0.0005 to 0.024
In motor vehicle exhausts	Trace
Intake, µg/day:	
Air, maximum	9.16
Food and water	116
Total intake, % from air[b]	7.9
Retained in lung, µg/yr	1.3
Maximum found in lung, µg	680
Minimum found in lung, µg	<10
Average total body content of V, µg	22
Soil, µg/g	100

[a] Principal source of vanadium is petroleum combustion products.
[b] At 20 m^3 inspired per day. Actually, about 75 percent is retained.

study, 7.9 percent of the daily intake of vanadium came from air at maximum urban levels. Among the 27 metals listed by Schroeder,[9] only mercury and lead showed a higher percentage intake via air than vanadium.

6.1.2.3. Medicinal Use — In the past, vanadium compounds have been prescribed infrequently for medicinal purposes. In the early 1900's, vanadium was used as a therapeutic agent for anemia, chlorosis, tuberculosis, and diabetes. It was also used as an antiseptic, as a spirochetocide, and as a tonic to improve appetite, nutrition, and general health. Sodium metavanadate was given by mouth in doses of 1 to 8 mg, and sodium tartrate was injected intramuscularly at levels as high as 150 mg. More recent studies on the effect of vanadium on blood cholesterol levels employed oral doses of soluble diammonium oxytartarovanadate for long periods to determine whether or not cholesterol levels could be lowered.[10,11] (Additional material on this subject will be found in Section 6.1.4).

6.1.3. Metabolism

6.1.3.1. Gastrointestinal Absorption — Vanadium salts are, in general, poorly absorbed from the human gastrointestinal tract. According to Curran et al.,[10] from 0.1 to 1 percent of 100 mg of vanadium in the form of the very soluble diammonium oxytartarovanadate was absorbed from the human gut. Within 24 hours, 60 percent of the vanadium absorbed was excreted via the kidneys. The remainder was retained in the liver and bone. It was mobilized rapidly from the liver and slowly from the bone following cessation of oral therapy.

In an unpublished study by Mouutain,[5] vanadyl sulfate was fed to adult male rats in daily doses of from 650 to 1,250 μg (160 to 310 μg vanadium). The mean absorption in this case was about 0.5 percent, with considerable variation as judged by urinary values.

6.1.3.2. Respiratory Deposition and Absorption — No data could be found that specifically describe the deposition of vanadium in the respiratory tract following inhalation. As with other particulate matter, deposition would be expected to be greatest in the submicron particle size fraction.

There is little doubt that vanadium can be absorbed following inhalation and deposition in the lung. However, the rates of pulmonary absorption have not been quantitatively determined, and no estimate has been made of the amount of vanadium that is coughed up, swallowed, and then possibly absorbed through the gastrointestinal tract. Lewis[12] reported that workers exposed to vanadium excreted four times as much vanadium in their urine as did controls.

Animal experiments have generally shown complete clearance of the relatively soluble vanadium pentoxide from the lung within 1 to 3 days following *acute* exposure.[4,13] Stokinger[5] has demonstrated, however, that some metal is present for a month or more following cessation of *chronic* exposure.

Levina[13] has reported that vanadium trioxide is cleared from the lung more rapidly than the pentoxide or ammonium vanadate following intratracheal instillation in rats. The fact that the pentoxide remains longest, he suggests, may be related to its "aggressiveness" or damage to the lung tissue.

In an autopsy study, Tipton and Shafer[14] have shown that vanadium accumulates in the lungs of the general population with age, reaching approximately 6.5 μg/g wet weight of tissue in those over age 65. The accumulation of vanadium with age was not observed in organs other than the lung.[14] This is not surprising, since vanadium is poorly absorbed from the gastrointestinal tract;[15] however, accumulation of vanadium in the lung suggests that some of the vanadium compounds deposited in the lung are relatively insoluble forms from contaminated air.[7]

6.1.3.3. Absorption Through the Skin — Stokinger[5] has reported that skin absorption occurs when a nearly saturated solution (20 percent) of sodium metavanadate is applied to rabbit skin. The dermal application of this compound on rabbits causes skin irritation. Human skin comes into contact with vanadium salts in the industrial environment, and skin sensitivity does develop.[4,16] However, the skin appears to be a minor route of uptake of the metal.

6.1.3.4. Transport — Although vanadium is transported in the serum, the carrier or "transvanadin" has never been isolated. Serum values for vanadium in 13 normal individuals ranged from 0.35 to 0.48 μg/ml (mean 0.42 μg/ml) in a study by Schroeder et al.[2] The vanadium was carried in the lipid fraction with none in the serum proteins. Schroeder et al. have estimated that a total of 1,380 μg of vanadium was circulating in the normal serum when samples were taken. They pointed out that this was approximately equivalent to the daily intake of patients on an institutional diet. This institutional diet apparently provided about ten times the usual dietary intake of vanadium. Vanadium was not detected in washed red cells from 19 control subjects, but five individuals taking diammonium oxytartarovanadate by mouth (4.5 mg V/day) had elevated vanadium levels in red cells (0.37 to 0.81 μg/ml, mean 0.48 μg/ml) with equivalent amounts in serum (mean 0.47 μg/ml). Thus it appears that excess vanadium in the serum spills over to be adsorbed or absorbed by the red cells. The data of Schroeder et al.[2] suggest that the serum carrier, if one exists, may be saturated or near saturated at levels (approximately 0.5 μg/ml) that may occur in the serum of individuals on institutional diets.

The influence of the oxidation state of intravenously injected compounds of vanadium-48 on uptake and distribution to selected organs and subcellular elements of liver in rats was studied by Hopkins and Tilton.[17] These investigators reported no significant differences in rate or amount of uptake of nanogram quantities of vanadium of three different oxidation states ($VOCl_3$, $VOCl_2$, and VCl_3). Hence, it appears that either oxidation state is not critical to transport, or that vanadium is somehow converted to a common oxidation state *in vivo*. Roshchin et al.[18] have found some evidence for partial conversion of vanadium trioxide to the pentavalent form in blood serum and in weakly acidic and basic solutions *in vitro*.

6.1.3.5. Distribution and Storage — In the rat studies by Hopkins and Tilton,[17] liver, kidney, spleen, and testes accumulated intravenously injected vanadium-48 for up to 4 hours and retained most of the radioactivity for up to 96 hours. By this time (96 hours), most other organs retained only 14 to 84 percent of their 10-minute uptake. After 96 hours, 46 percent of the vanadium-48 had been excreted via the urine and 9 percent via the feces. Vanadium-48 that was retained in the liver was observed over the first 96 hours to decrease in the supernatant fraction of centrifuged liver homogenate from 57 to 11 percent of total liver radioactivity. Vanadium-48 in the mitochondrial and nuclear fractions increased over the first 96 hours from 14 to 40 percent of the total. Radioactivity in the microsomal fraction remained relatively constant.

Soremark et al.[19] reported highest uptake of vanadium-48 from $^{48}V_2O_5$ in young rats in rapidly mineralizing areas of dentin and bone. In pregnant females, they noted a concentration of radiovanadium in the fetus as well as in maternal bones and teeth. Thus it is important to note the potential exposure of the unborn offspring to vanadium via the maternal-fetal circulation. In another study, Soremark and Üllberg[20] demonstrated high uptakes in mice in mammary glands, liver, renal cortex, and lung.

In $^{48}VOCl_3$ retention studies in rats, Strain et al.[21] found that hair retention correlates with retention in aorta, bone, and liver, although levels in hair are much lower than in the three other tissues. Hair retention did not correlate with blood retention. Blood levels were undetectable after 20 days. Bone retention was highest in young rats of both sexes.

According to Schroeder et al.,[2] storage of available vanadium in man is mainly in fat and serum lipids (see Section 5.2.5 on body burdens and animal tissue levels). Large amounts of vanadium are also reported in crude fat from beef, pork, and lamb. Keratin has been suggested as a minor storage depot.[2]

In general, animal tissues are low in vanadium. None was found in lungs of several animals, in contrast to the previously mentioned[14] accumulation in the lungs of man. Soremark[22] has reported values ranging from less than 1 to several parts per billion (wet weight). Hopkins and Mohr[23] found similar levels in heart, kidney, and liver tissues from chicks on a vanadium-deficient diet. However, on a normal, supplemental diet, vanadium levels were 10 times higher. The report by Hopkins and Mohr[23] indicated that vanadium is an essential element for chicks; its deficiency resulted in reduced feather growth and lowered blood cholesterol. Strasia[24] and Schwarz and Milne[25] also found vanadium to be essential in the rat, with deficiency reducing growth rate. Amounts of vanadium required both for chicks and rats are those normally found in nutrients and tissues, however.

6.1.3.6. Excretion — Vanadium that is absorbed is excreted mainly in urine but also in feces. In long-term balance studies, Tipton et al.[8] demonstrated that 1 percent of dietary vanadium is excreted in the urine (Table 6.3). Vanadium was not detected by two different methods of analysis in urines of 36 normal subjects.[8] However, when 4.5 to 9.0 mg of vanadium as diammonium oxytartarovanadate was fed daily to 16 elderly persons, urinary excretion, although quite variable, amounted to a mean of 5.2 percent of the amount ingested.[8]

Table 6.3. EXCRETION OF VANADIUM BY THREE HUMAN SUBJECTS[8],[a]

Excretion route	Subject C		Subject D		Subject E	
	μg	% of Total	μg	% of Total	μg	% of Total
Feces	120 ∓ 3.0	86	150 ± 2.0	83	37 ± 0.5	75
Urine	18 ± 0.2	13	23 ± 0.2	13	12 ± 0.2	24
Sweat, hair, etc.[b]	—	1	—	4	—	1
Total	140 ± 3.0	100	180 ± 2.0	100	49 ± 0.5	100

[a] Mean values given. Subjects C and D were studied for 347 days; subject E for 70 days.
[b] Inferred values to yield 100%.

A study by Dimond et al.,[26] in which ammonium vanadyl tartrate was fed to young and middle-aged patients, demonstrated that urinary excretion was unpredictable relative to oral dosage. These investigators suggested that variable absorption was the reason for wide fluctuations in urinary excretion. Jaraczewska and Jakubowski[27] also concluded that concentrations of vanadium pentoxide in air in industrial exposures could not, with certainty, be correlated with vanadium concentrations in urine. However, the latter pointed out the similarity in urinary clearance of intravenously injected and tracheobronchially administered sodium vanadate in guinea pigs.

Since most of the ingested vanadium is not absorbed, the preponderance of vanadium elimination is via the feces. Data from Tipton et al.[8] on three normal individuals (Table 6.3) showed that an average of 81 percent of ingested vanadium was excreted via the feces, compared to 17 percent via the urine.

6.1.3.7. Homeostasis — The relatively high serum levels (0.35 to 0.48 μg/ml) in normal individuals and low or undetectable urine levels reported by Schroeder et al.[2] suggest that vanadium in serum is not filtered by the renal glomeruli. Alternatively, renal tubular reabsorption may be very efficient. Filtration or reduced reabsorption may function to eliminate vanadium when the ability of serum lipids or lipoproteins to bind vanadium is exceeded. Estimates of intestinal absorption following the administration of soluble vanadium compounds seem to indicate a proportional reduction in absorption with increasing dose. In any case, it appears that homeostatic mechanisms are operative to maintain consistently low tissue levels of vanadium.

6.1.4. Human Health Effects

6.1.4.1. Respiratory Effects of Human Industrial Exposure — Most of the clinical symptoms observed following industrial exposures to vanadium reflect its irritative effects on the upper respiratory tract. Dutton [28] is believed to be the first to have described the health effects of industrial exposure to vanadium-bearing ores. Dutton reported a dry, paroxysmal cough with hemoptysis, and irritation of the eyes, nose, and throat. A temporary increase in hemoglobin and red cells was followed by a reduction in both and the onset of anemia. Vanadium was recovered in all bodily secretions. *Post mortem* examination revealed highly congested lungs with destruction of the alveolar epithelium, and congested kidneys with evidence of hemorrhagic nephritis. Unfortunately, the workers studied frequently suffered from pulmonary tuberculosis, which undoubtedly produced many symptoms that were aggravated by vanadium exposure. Also, Dutton did not provide details as to the number of workers examined or the incidence of the symptoms he described.

A subsequent observation by Symanski[3] of relatively healthy metal workers exposed to vanadium pentoxide dust revealed severe conjunctivitis, rhinitis, pharyngitis, chronic productive cough, and tightness of the chest. X-Rays demonstrated bronchitis, and Symanski expected bronchiectasis with longer exposure. Symanski's report differed from Dutton's[28] in that the former found no evidence for a generalized systemic action of vanadium.

Rundberg[29] observed bronchitis with purulent sputum, general weakness, and skin irritation of the face and hands of 20 men handling vanadium pentoxide in a metallurgical works. Balestra and Molfino[30] reported productive cough, bronchitis, and shortness of breath in 25 workers exposed to vanadium pentoxide dust from petroleum ash. Other substances were involved, but chest X-rays showed definite lung markings suggesting pneumoconiosis. Bronchiectasis was suspected in two cases.

Wyers[31,32] described his supervision of 50 to 90 workers exposed to vanadium pentoxide as an oil combustion residue and to slag from production of ferrovanadium. Findings included bronchospasm, often with elevated blood pressure and an accentuated pulmonary second sound, a paroxysmal cough, dyspnea, skin pallor, tremor of fingers, palpitation, chest pains, and reticulation of the lungs. Thus, Wyers emphasized the irritant effects of vanadium pentoxide on the respiratory tract but also found some evidence of systemic toxicity. An indication of exposure to vanadium first reported by Wyers was "green tongue," believed to result from reduction of vanadium pentoxide to the trioxide in the mouth. Data in Wyers' report are limited to 10 case histories (that is, occurrence in one or two of 10 men observed), and no control group was examined.

In 1950, Sjøberg[4] published an extensive report with data on the dust content of the air in a metallurgical plant producing vanadium pentoxide. The dust particles were relatively large in size — 39 percent less than 12 μm, 22 percent less than 8 μm — and it was estimated that 6.5 mg V_2O_5/m^3 represented the worst exposure condition. Of thirty-six men between 20 and 50 years of age who had been employed in the plant 4 years, 22 had a dry cough, wheezing sounds could be detected in 31, and 27 were short of breath. One man developed acute pneumonitis, and four others developed bronchopneumonia. There was no concrete evidence of systemic toxicity.

A dry eczematous dermatitis developed in nine men in Sjøberg's[4] study, but only one man showed a positive patch test. Sjøberg and Ringer[33,34] believed that allergy might play a role in the development of eczema and pneumonitis following vanadium exposure. Zenz et al.[35] also considered this an explanation for more severe symptoms found on reexposure in their study.

Six years later, in a follow-up to his 1950 study, Sjøberg[34] reported that the 16 men most severely affected still complained of shortness of breath, coughing, fatigue, and wheezing; two still had bronchitis. However, spirometric measurements, cardiac function tests, electrocardiograms, hematological tests and urinalyses were essentially normal.

Bronchitis and conjunctivitis resulting from exposure to soot (containing 6 to 11 percent vanadium) in cleaning the stacks of oil-fired boilers were first recognized by Frost.[36] He reported no systemic symptoms, but a subsequent report of a boiler-cleaning operation by Williams[37] noted secondary symptoms of lassitude and depression. Within 0.5 to 21 hours of exposure primary symptoms were sneezing, nasal discharge, lacrimation, sore throat, and substernal pain. Within 6 to 24 hours, secondary symptoms developed, consisting of dry cough, wheezing, labored breathing, lassitude, and depression. In some cases, the cough became paroxysmal and productive. Symptoms lessened only after removal from the working environment for 3 days. Air sampling showed that most dust particles were smaller than 1 μm. The vanadium concentration ranged from 17.2 mg/m^3 in a superheater chamber to 58.6 mg/m^3 in a combustion chamber (Tables 6.4 and 6.5).

Other observations of boiler-cleaning operations were made by Fallentin and Frost,[38] Sjøberg,[39] Thomas and Stiebris,[40] Hickling,[41] Roshchin,[42] and Troppens.[16] The latter paper describes the symptoms as a slight cold or flu condition that ended in bronchitis. Following recovery, the workers were tired, debilitated, irritable, and without appetite They also complained of watery eyes such as might occur with a slight case of conjunctivitis. Troppens[16] describes the first symptoms as swelling of face and eyes as early as 20 minutes after entering the boiler area. Removal from exposure for 2 to 3 weeks results in disappearance of symptoms. Skin blemishes described as allergic dermatoses were attributed to absorption of vanadium through sensitive skin. Vanadium in the urine was elevated one-and-one-half to threefold. Mention was made of increased susceptibility of the vanadium worker to asthmatic bronchitis and emphysema, twice as high as other workers according to data derived from Koelsch and cited by Troppens.[16]

Table 6.4. THERMAL-PRECIPITATOR SAMPLES TAKEN FROM SUPERHEATER DURING CLEANING OPERATION[37]

Diameter of particles, μm	No. of particles		Concentration of particles	
	per ml of air	no %	mg/m^3 of air	wt. %
0.15 to 1.0	3300	93.6	0.36	2.9
1.1 to 5.5	217	6.14	4.09	33.3
5.5 to 11	9	0.26	7.85	63.8

Table 6.5 ANALYSIS OF DUST FROM BOILER DURING CLEANING[37]

Location	Dust concentration, mg/m^3 of air	Vanadium concentration, % of dust	Vanadium concentration, mg/m^3 of air
Superheater chamber	659	6.1	40.2
Superheater chamber	239	7.2	17.2
Combustion chamber	489	12.7	58.6

With all reports of respiratory symptoms relating to boiler-cleaning operations it must be remembered that sulfates and sulfuric acid are also present in boiler soot and may be in part responsible for irritative effects. Hudson[1] has suggested that quick onset of symptoms (lacrimation with nose and throat irritation) with rapid recovery following removal from exposure is characteristic of exposure to sulfur oxide gases. Dermatitis is said to be characteristic of exposure to acid sulfates. Response to vanadium exposure is characterized by some delay in the onset of irritative symptoms (a few hours to several days), with persistance of symptoms following removal from exposure.

Additional reports have appeared relating to the health effects of occupational handling of pure vanadium pentoxide or vanadate dusts. Among them are reports by Pielsticker,[43] Gulko,[44] Matantseva,[45] and Zenz et al.[35] Zenz et al.[35] described a uniform acute illness that occurred in 18 workers pelletizing pure vanadium pentoxide; it was characterized by a rapidly developing mild conjuctivitis, severe pharyngeal irritation, a nonproductive persistent cough, diffuse rales, and bronchospasm. With severe exposure, four men complained of itching skin and sensation of heat in the face and forearms. The symptoms became more severe after each exposure, which suggested a sensitivity reaction; however, duration of symptoms was not prolonged by the subsequent exposures.

Studies concerned primarily with mining, milling, and smelting operations have been published by Vintinner et al.,[46] Lewis,[12] Rajner,[47] and Roshchin.[48] The investigation of Lewis[12] is particularly significant in that the maximum exposure was only 0.925 mg V (as V_2O_5)/m^3 of air, and in most cases 0.3 mg V/m^3 was the exposure level. More than 92 percent of dust particles were smaller than 0.5 μm in every process area sampled. Symptoms of cough with sputum production, eye, nose, and throat irritation, and wheezing were related to physical findings of wheezes, rales, or rhonchi, injected pharynx, and green tongue. All of these symptoms and physical findings were statistically significant as compared to controls (Table 6.6 and 6.7). The report by Rajner[47] on 30 vanadium workers in a metallurgical plant in Czechoslovakia describes particularly severe symptoms, but gives no estimates of exposure except in conjunction with urinary vanadium levels. In acutely affected workers, vanadium values were about 4,000 μg/liter of urine. The average values among permanent employees was 45 μg/liter, but among vanadium pentoxide smelter workers, the average was 400 μg/liter. When a new production process was introduced, symptoms of acute vanadium intoxication occurred in these workers that included severe respiratory difficulties, headaches, dejection, and loss of appetite after 16 hours of work. Acute inflammatory changes of the upper respiratory tract with copious mucus production, edema of the vocal cords, and profuse nose bleeding were reported. Workers who had been exposed for up to 22 years (27 of the workers), mostly in ferrovanadium and vanadium pentoxide smelting operations, complained of coughing and eye, nose, and throat irritation (all 27 workers), breathing difficulties during physical exertion (more than 14 cases), and headaches (12 cases). Clinical findings included intensive hyperemia of the mucosa of the nasal septum in 20 workers and perforation of the nasal septum in four workers. Intensive hyperemia of the mucosa of the throat and larynx with dilated fine capillaries was found in half of the workers. Bronchoscopy indicated the presence of chronic bronchitis, and bronchial smears revealed sloughed epithelium. "Slight (pulmonary) functional disorders and beginning emphysema" were reported in five cases. Pneumoconiosis was not detected on X-ray, and no heart changes or alterations in blood chemistry were reported.

Table 6.6. SYMPTOMS IN VANADIUM WORKERS[12]

Symptom	Incidence, %		X^2 value
	Control	Exposed	
Cough	33.3	83.4	13.71[a]
Sputum	13.3	41.5	5.55[b]
Exertional dyspnea	24.4	12.5	5.592
Eye, nose, throat irritation	6.6	62.5	23.17[a]
Headache	20.0	12.5	0.124
Palpitations	11.1	20.8	0.538
Epistaxis	0	4.2	0.148
Wheezing	0	16.6	5.20[b]

[a] Significant beyond $p = 0.01$.
[b] Significant at $p = 0.02$.

Table 6.7. PHYSICAL FINDINGS IN VANADIUM WORKERS[12]

Physical finding	Incidence, %		X^2 value
	Control	Exposed	
Tremors of hands	4.5	4.2	0.320
Hypertension	13.3	16.6	0.0002
Wheezes, rales, or rhonchi	0.0	20.8	6.93[a]
Hepatomegaly	8.9	12.5	0.003
Eye irritation	2.2	16.6	2.94
Injected pharynx	4.4	41.5	12.62[a]
Green tongue	0.0	37.5	14.53[a]

[a] Significant beyond $p = 0.01$.

Other occupational areas in which respiratory effects of vanadium exposure have been reported include operations connected with the gasification of fuel oil (Fear and Tyrer[49]) and with the maintenance of gas turbines (Browne[50]).

A recurrent inadequacy of all of these reports on industrial exposure to vanadium is the failure to consider or evaluate the influence of smoking on the clinical findings.

In summary, the consensus of industrial hygienists is that there is insufficient evidence to support the view that vanadium, except at extremely high concentrations, causes generalized systemic toxicity. However, extensive evidence exists that vanadium dust (usually the pentoxide) is severely irritating to the mucous membranes of the eyes, nose, throat, and respiratory tract. Bronchitis and bronchospasm are characteristic symptoms, and pneumonia occasionally develops. Chronic productive cough and wheezing persist even after the subject is removed from exposure. Many investigators have reported that vanadium workers are more susceptible to colds and other respiratory illnesses.[16,18,51,52] Recent studies by Waters et al.[53,54] have demonstrated that vanadium oxides are very toxic for rabbit alveolar macrophages *in vitro*. Toxicity is related to the availability of soluble vanadium. In view of the central role of the alveolar macrophage in pulmonary defense, these studies suggest that exposure to vanadium may impair the lung's resistance to secondary respiratory infection. Thus, an attractive hypothesis to account for these reports and for many of the chronic respiratory symptoms is that vanadium exposure predisposes the individual to secondary respiratory infection. However, though there is no evidence to the contrary, a chemico-bacterial basis for the respiratory symptoms observed in industrial workers remains to be demonstrated.

6.1.4.2. Occupational Exposure Limits — As defined by The American Conference of Governmental Industrial Hygienists,[55] threshold limit values (TLVs) "refer to airborne concentrations of substances and represents conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect. . . . Threshold limit values refer to time-weighted concentrations for a 7- or 8-hour workday and 40-hour workweek."

In 1961, the American Conference of Governmental Industrial Hygienists adopted TLVs for vanadium compounds. In 1971, on the basis of additional data, new TLVs were established.[56] Finally, in 1972, the TLVs were revised upward in that concentrations were expressed as vanadium rather than vanadium pentoxide; however, the value for vanadium pentoxide fume was designated a ceiling concentration. (A ceiling value should not be exceeded and is, in effect, a maximum allowable concentration.) Table 6.8 lists TLVs for vanadium compounds for 1961, 1971, and 1972.

An outline of the industrial hygiene literature cited in the *Documentation of Threshold Limit Values*,[57] is given in Table 6.9 These data, along with the experimental studies of Roshchin,[60] Stokinger,[61] and Zenz and Berg,[62] constitute the scientific evidence upon which the TLVs were based.

Table 6.8. THRESHOLD LIMIT VALUES FOR VANADIUM COMPOUNDS, 1961, 1971, 1972[55,56]

Compound	Threshold limit value, mg/m^3
1961:	
Vanadium pentoxide (dust)	0.5
Vanadium pentoxide (fume)	0.1
Ferrovanadium (dust)	1.0
1971:	
Vanadium pentoxide (dust) at V_2O_5	0.5
Vanadium pentoxide (fume) V_2O_5	0.05
1972:	
Vanadium pentoxide (dust) as V	0.5
Vanadium pentoxide (fume) as V	0.05 (ceiling)

Table 6.9 DOCUMENTATION OF THRESHOLD LIMIT VALUES: OUTLINE OF LITERATURE FINDINGS ON INDUSTRIAL EXPOSURES TO VANADIUM[a]

Industry	Investigator and reference	Vanadium compound	Concentration, mg/m³	Symptoms
Vanadium refinery	Sjøberg[1]	V_2O_5	$\leqslant$ 12	Mild respiratory irritation
Boiler cleaning	Sjøberg[39]	$V_2O_5(V_2O_3)$	2 to 85	Respiratory irritation
Boiler cleaning	Williams[57]	$V_2O_5(V_2O_3)$	30 to 104	Intoxication (used respirators to some extent)
Vanadium ore mining and processing	Vintinner et al.[46]	V_2O_5	3 to 100	Local respiratory effects, no systemic poisoning
Boiler cleaning	McTurk et al.[58]	V_2O_5	99	No intoxication (gauze filters worn)
V_2O_5 processing	Gulko[44]	(V_2O_5)	0.5 to 2.2	Eye and bronchial irritation
Vanadium refinery	Lewis[12]	(V_2O_5)	0.2 to 0.5	Respiratory irritation
Phosphor plant	Tebrock and Machle[59]	Yttrium europium vanadate	1.5 (as V_2O_5)	Conjunctivitis, tracheobronchitis, and dermatitis
Vanadium refinery	Hudson[1]	V_2O_5 and NH_4VO_3	0.25	Green tongue, metallic taste, throat irritation and cough

[a]Literature findings are cited in Documentation of Threshold Limit Values.[57]

The 1972 TLV for vanadium pentoxide dust and fume is compared with TLVs for some other metallic oxides in Table 6.10.

Table 6.10. COMPARISON OF 1972 TLV FOR VANADIUM PENTOXIDE DUST AND FUME WITH THRESHOLD LIMIT VALUES FOR OTHER METALLIC OXIDES[60]

Compound	Threshold limit value, mg/m^3
Boron oxide	10
Cadmium oxide (fume)	0.1 (ceiling)
Calcium oxide	5
Iron oxide (fume)	10
Magnesium oxide (fume)	10
Osmium tetroxide	0.002
Vanadium pentoxide (fume) as V_2O_5	0.05 (ceiling)
Zinc oxide (fume)	5

The TLVs for vanadium have been established mainly on the basis of the irritative effects of vanadium compounds on the respiratory mucosae. Schroeder,[7] after Stokinger,[5] has ascribed the irritative effects of vanadium pentoxide to the acidity of its aqueous solutions. Vanadium trichloride is perhaps the most potent irritant, followed by vanadium pentoxide, ammonium and sodium vanadate, vanadium trioxide, and vanadium alloys, in that order. These irritative effects are tolerated by workers since they are not accompanied by pain and, except in severe exposure, do not develop rapidly.

6.1.4.3. Respiratory Effects of Human Experimental Exposure — The study of Zenz and Berg[62] involved nine healthy volunteers, aged 27 to 44, who had previously submitted to lung function tests for the purpose of developing baseline data. Two of the volunteers, exposed to vanadium pentoxide dust at 1 mg/m^3 for 8 hours, developed sporadic coughing after 5 hours and frequent coughing near 7 hours. Coughing lasted 8 days, but lungs remained clear; and there were no other signs of irritation. Lung function tests, complete blood counts, urinalyses, and nasal smears were normal for up to 3 weeks. After this time, the same two volunteers were accidently exposed to a "heavy cloud" of vanadium pentoxide dust for 5 minutes. A productive cough developed within 16 hours, and within 24 hours, rales and expiratory wheezes developed throughout the lung. Pulmonary function remained normal. Isoproterenol (1:2000) relieved the symptoms for about an hour. Then coughing began again and continued for 7 days. There were no other symptoms. Eosinophils were not present in nasal mucus.

In the next test, the exposure concentration was reduced to 0.2 mg/m^3 ($\pm$ 0.05 standard deviation) for 8 hours. Light microscopy indicated that 98 percent of the particles were smaller than 5 μm. Again, by the following day, all five men exposed had developed a loose cough. Coughing, without other systemic symptoms, persisted for 7 to 10 days. Pulmonary function tests and differential white blood counts remained normal. Vanadium in the urine was highest (0.013 mg/100 ml or 0.13 μg/ml) on the third day, with none detectable after 7 days. Maximal fecal vanadium was 3 μg/g, with none detectable after 14 days.

Finally, two volunteers were exposed to 0.1 mg/m^3 of vanadium pentoxide (0.056 mg V/m^3) for 8 hours. Within 24 hours, considerable mucus had formed. The mucus was cleared by slight coughing, which became more severe after 48 hours, subsided after 72 hours, and disappeared after 96 hours. Zenz and Berg described the symptoms as a "distinct clinical picture of pulmonary irritation"[62] despite the fact that pulmonary function tests and other clinical findings remained normal. All of the individuals exposed in the study by Zenz and Berg developed acute symptoms of marked pulmonary irritation upon initial exposure to vanadium pentoxide dust. Two individuals exposed a second time to the dust at 0.1 mg/m^3 (0.056 mg V/m^3) developed the irritative symptoms. Hence, the TLV for vanadium pentoxide dust and fume was lowered to 0.05 mg V/m^3. It is likely, however, that even at the new TLVs healthy individuals will suffer symptoms of respiratory irritation. Furthermore, individuals with a history of respiratory disorders may be expected to experience distress before TLVs are reached.

6.1.4.4. Diagnosis of Human Exposure — The respiratory symptoms resulting from vanadium exposure are so similar to those of acute infection of the respiratory tract that unequivocal diagnosis is difficult. Certain biochemical indices, along with evidence of probable exposure, can assist in diagnosis, but no specific test is recommended. Determination of the vanadium content in blood and urine provides definitive qualitative documentation of exposure. In view of the work of Schroeder,[7] it would seem desirable to measure the vanadium content of the serum separately from that of the cellular elements, since the concentration of vanadium in the latter may be more indicative of exposure levels. A decreased urinary output of ascorbic acid may be one characteristic of vanadium exposure as reported by Watanabe et al.,[63] although differences from controls do not appear great enough to make the test useful clinically.

The most sensitive test developed to date involves measurement of the cystine content of the fingernails. This test was correlated to vanadium exposure in workers and has provided a useful clinical tool in the management of health of vanadium workers.[64] A decrease in cystine in fingernails was demonstrated when urinary vanadium levels were only 0.02 to 0.03 μg/ml. A similar reduction in the cystine content of rat hair was also reported[65] when vanadium in the diet ranged from 25 to 1,000 ppm. There is some evidence to suggest that vanadium may directly inhibit the synthesis of cystine or cysteine.[64,65] Also, these reductions in cystine content in nails and hair may be related to excretion of cystine in the urine (cystinuria) since an increased neutral sulfur fraction which is indicative of a cystinuria, is observed in the urine of rats fed vanadium.[66] Cystinuria is known to be associated with Wilson's disease, in which there is a genetically determined lack of the copper transport protein, ceruloplasmin — a plasma globulin. A possible implication of increased cystine excretion in the urine would be that vanadium somehow interferes with copper metabolism. Keenan[67] noted that in spectographic analyses of livers of animals exposed to vanadium, the intensity of copper lines diminished as vanadium intensity increased. Conversely, deJorge et al.[68] have demonstrated decreased serum levels of vanadium and increased serum levels of copper and ceruloplasmin in Charcot-Marie muscular atrophy. These changes were proportional to symptom severity.

6.1.4.5. Effects of Community Exposure — In 1960, Stocks[69] reported the results of a statistical study in which airborne concentrations of 13 trace elements were correlated with mortality from lung cancer, pneumonia, and bronchitis in 23 localities in Great Britain. Beryllium and molybdenum proved to correlate best, whereas arsenic, zinc, and vanadium showed weak associations with mortality from lung cancer. After eliminating beryllium, molybdenum, and social index (taking into consideration population density, sex, and age), vanadium retained a coefficient of correlation with lung cancer of 0.347. With regard to mortality from pneumonia in the localities studied, beryllium was an important element in both sexes. Vanadium was also correlated with mortality from pneumonia, but only in males. When social index and beryllium were held constant, a significant coefficient of correlation with mortality from pneumonia (0.443) remained for vanadium. Molybdenum was strongly correlated with mortality involving bronchitis in both sexes. When social index and molybdenum were eliminated, vanadium gave a coefficient of correlation with mortality involving bronchitis of 0.563. Beryllium, molybdenum, and vanadium also showed associations with mortality from cancers other than of the lungs in males, but not in females.

Another statistical study by Hickey et al.[70] considered 10 metals in the air, including vanadium, in 25 communities in the United States. Various techniques, including canonical analysis, were used to correlate airborne metal concentrations with mortality indices (1962 and 1963) involving eight disease categories. The incidence of several diseases, including "diseases of the heart," nephritis, and "arteriosclerotic heart" could be correlated reasonably well with air levels of vanadium, cadmium, zinc, tin, and nickel. The addition of vanadium to cadmium produced a reduction of more than 10 percent (the greatest reduction) in the error of variance. A high intercorrelation between vanadium and nickel was unexplained.

These studies of Stocks and of Hickey are exploratory in nature. The relationships disclosed cannot be considered to be causal in nature without further study. The Hickey study is of a very preliminary nature, with no adjustments for the basic pertinent variables normally employed. The Stocks investigation is more extensive, considering a number of important adjustments (e.g., age, sex) and various interactions such as other metals and social factors. Other pollutions — smoking, for example — have not been examined. Intercorrelations were not fully explored. An additional multivariate analysis of air vanadium levels in relation to selected white male mortality levels is contained in an unpublished EPA staff study by Pinkerton et al.[71] Several categories of cardiovascular disease were used, and also influenza-pneumonia. Vanadium was not correlated with the latter, but was with the cardiovascular categories. However, adjustments for population density produced considerable reduction in some of these relationships. The authors' comment was: "These results suggest that the observed statistical associations of air manganese and air vanadium were not causal associations, and represented either a reflection of other more directly associated causes or statistical artifacts."[71] Another difficulty with interpretation of these observations lies in their differences from observations on health effects seen in vanadium workers. Additional occupational and population studies on chronic illness in relation to vanadium exposure are needed to determine whether some consistency of findings and evidence of a dose-response relationship exist.

6.1.4.6. Other Effects of Human Exposure — As evidenced by the very high levels of vanadium prescribed medicinally at the turn of the century (see Section 6.1.2.), it is well established that the metal is not very toxic for man when ingested or when administered intramuscularly. No adverse health effects have been reported from ingestion of vanadium at any levels normally found in food or water. However, it has been estimated that a lethal dose for a 70-kg (154-lb) man would be 30 mg V_2O_5 (0.43 mg V_2O_5/kg) if introduced directly into the circulation in soluble form.[1]

Vanadium has been prescribed in recent years only in experimental investigations of its effects on circulating cholesterol levels. In 1959, Curran et al.[10] conducted a clinical study in which five normal adult males were fed 150 to 200 mg/day soluble diammonium oxytartarovanadate (21 to 30 mg V/day) for a 6-week period. At the end of this time, plasma cholesterol was significantly reduced. Lewis[72] conducted a study of 32 vanadium workers and 45 controls; ages were matched in the two groups, and all persons were over 40 years old. The vanadium workers were observed to excrete greater than normal amounts of vanadium and exhibited slightly lower serum cholesterol levels than controls. Mean control cholesterol values were 230.9 and 226.7 mg/100 ml. Levels in the vanadium workers were less by 26 and 20 mg/100 ml ($p < 0.05$). A clinical study by Sommerville and Davies[11] of 12 patients (nine of whom were hypercholesterolemic) given diammonium vanadotartrate showed no significant changes in serum cholesterol levels over 5.5 months. The mean pretreatment control level of serum cholesterol was 411 mg/100 ml, and the mean age was 49.2 years. Hence, in addition to being hypercholesterolemic, these individuals were older than those studied by Curran et al.[10] Dimond et al.[26] observed temporary drops (not statistically significant) in cholesterol in two of six patients given ammonium vanadyl tartrate for several weeks at levels of 50 to 100 mg/day. No statistically significant changes were observed in blood lipids, phospholipids, triglycerides, 17-ketosteroids, or 17-hydroxycorticosteroids. The subjective symptoms of fatigue and lethargy were present in two patients while they were taking vanadium. All complained of cramps and loosened stools, and all developed "green tongue." Schroeder et al.[2] reported that their findings were similar to those of Dimond et al.[26] and expressed the view that the slight effects of vanadium on serum cholesterol were pharmacological and not caused by correction of a physiological deficiency. They pointed out that dietary regimens based on the consumption of unsaturated fats, which lower plasma cholesterol in man, are associated with the intake of 1 to 4 mg vanadium per day and that the feeding of vanadium-poor, saturated fats raises cholesterol. Hence, they note the interesting possibility that the ratio of vanadium to fat may be a factor in the homeostasis of circulating cholesterol.

6.1.5. Effects on Experimental Animals

6.1.5.1. General Toxicity — The toxicity of vanadium salts varies among mammals and according to routes of administration (Table 6.11).[2,7,73] The smaller animals, including the rat and mouse, tolerate the metal fairly well. The rabbit, horse, and man are more sensitive.[1] In general, toxicity is low when the metal is given by the oral route, moderate by the respiratory route, and high by injection. Lethal doses for various vanadium salts injected intravenously in rabbits and subcutaneously in guinea pigs, rats, and mice are shown in Table 6.12. The toxicity of vanadium also varies considerably with the nature of the compound, although vanadium is toxic both as a cation and as an anion. As a general rule, toxicity increases as valency increases, pentavalent vanadium being the most toxic. Among the oxides of vanadium, the pentavalent vanadium pentoxide is more soluble and more toxic than the less common trioxide or dioxide.

Table 6.11. TOXICITY OF VANADIUM SALTS TO MAMMALS[7,7,8]

Method and duration of administration	Type of mammal	Valence	Amount	Effect
Oral (in diet), months	Rat	5	1,000 ppm	Severe
Oral (in diet), life	Rat	5	100 ppm	Moderate
Oral (in water), life	Rat	5	8 ppm	None
Inhalation, 2 hr daily, months	Rat	3	40 to 70 mg/m^3	Severe
	Rabbit	3	40 to 70 mg/m^3	Severe
	Rat	5	18 mg/m^3	Severe
	Rabbit	5	18 mg/m^3	Severe
	Man	—	1 to 50 mg/m^3	Slight
	Man	—	33 to 66 mg/m^3	Lethal (estimated)
Parenteral intraperitioneal	Rat	3	4 to 5 mg/m^3	Lethal
Intravenous acute	Rat	5	6 mg/kg	Lethal
	Rabbit	5	1 to 20 mg/kg	Lethal
	Cat	5	7.18 mg/kg	Lethal
	Man	5	16.8 mg/km	Lethal (estimated)

Table 6.12. LETHAL DOSES OF SELECTED VANADIUM SALTS[1] (mg V_2O_5/kg)

Vanadium salt	Rabbit[a]	Guinea pig	Rat	Mouse
Colloidal vanadium pentoxide	1 to 2	20 to 28	—	87.5 to 117.5
Ammonium metavanadate	1.5 to 2.0	1 to 2	20 to 30	25 to 30
Sodium orthovanadate	2 to 3	1 to 2	50 to 60	50 to 100
Sodium pyrovanadate	3 to 4	1 to 2	40 to 50	50 to 100
Sodium tetravanadate	6 to 8	18 to 20	30 to 40	25 to 50
Sodium hexavanadate	30 to 40	40 to 50	40 to 50	100 to 150
Vanadyl sulfate	18 to 20	34 to 45	158 to 190	125 to 150
Sodium vanadite	—	30 to 40	10 to 20	100 to 150

[a] Rabbits were injected intravenously; other animals, subcutaneously.

According to Roshchin,[51] the toxic effects of vanadium compounds in experimental animals are highly specific; however, at the present time, the mechanisms of these effects are incompletely defined. In the sections that follow, an attempt is made to integrate the experimental data presently available so as to suggest plausible mechanism for the observed effects.

6.1.5.2. Respiratory Effects — A number of studies have dealt with respiratory exposure to vanadium pentoxide (Table 6.13[4,44,74,75]). Sjøberg[4] has reported in great detail experiments in which rabbits were exposed to vanadium pentoxide dust particles, nearly all of which were smaller than 10 μm in diameter. High concentrations over *short periods* of time were quite toxic; 205 mg V_2O_5/m^3 (or 115 mg V/m^3) was lethal in 7 hours. At these levels, tracheitis was marked and was accompanied by pulmonary edema and bronchopneumonia. Conjunctivitis, enteritis, and fatty infiltration of the liver were also observed. Vanadium was detected in ashed lung, liver, kidney, and intestine.

Sjøberg[4] also carried out *long-term* studies in which rabbits were exposed to 20 to 40 mg V_2O_5/m^3 (or 11 to 22 mg V/m^3) intermittently for 1 hour each day for several months. Upon sacrifice of the animals, pathological changes observed included chronic rhinitis and tracheitis, emphysema, patches of lung atelectasis, and bronchopneumonia. Pyelonephritis was seen in some cases. Vanadium was detected in ashed lung liver and kidney, but not, as with heavy exposure, in the intestine. There were no fibrotic changes or specific chronic lesions in the lungs, nor was there a visible accumulation of particles. These findings, plus the presence of vanadium in the liver and kidney, were evidence of rapid clearance and/or absorption from the lung.

Gulko[44] showed that *continuous* exposure of rabbits to 10 to 30 mg V_2O_5/m^3 (5.6 to 16.8 mg V/m^3) was toxic to the animals and caused bronchitis, pneumonia, loss of weight, and bloody diarrhea.

Roshchin[74] described the results of *acute* inhalation studies using rats. Vanadium pentoxide was administered at 10 to 70 mg/m^3 as the condensation aerosol (fume) or 80 to 700 mg/m^3 as the grinding aerosol (dust); ammonium vanadate was given (presumably as the grinding aerosol) at 1,000 mg/m^3; and ferrovanadium was given as the grinding aerosol at 1,000 to 10,000 mg/m.[3] The minimum concentration of vanadium pentoxide (in the form of a condensation aerosol) that gave rise to mild signs of acute poisoning was 10 mg/m^3 of air. The obsolute lethal concentration for the condensation aerosol was 70 mg/m.[3] Grinding aerosols (containing large particles) were only one-fifth as toxic as condensation aerosols. Inhalation of grinding aerosols of ferrovanadium did

Table 6.13. RESPIRATORY EFFECTS OF VANADIUM PENTOXIDE IN EXPERIMENTAL ANIMALS

Investigator and reference	Animal	Form	Concentration, mg/m^3	Exposure time	Pathological findings
Sjøberg[4]	Rabbit	Dust	205	7 hr	Conjunctivitis, tracheitis, plumonary edema, bronchopneumonia, enteritis, fatty liver, death
Sjøberg[4]	Rabbit	Dust	20 to 40	1 hr/day, for several months	Chronic rhinitis, tracheitis, emphysema, atelectasis, bronchopneumonia, pyelonephritis
Gulko[44]	Rabbit	Dust	10 to 30	Continuous, acute	Bronchitis, pneumonia, weight loss, bloody diarrhea
Roshchin[74]	Rat	Dust, fume	80 to 700 10 to 70	Continuous, acute	Hemorrhagic inflammation in lungs, hemorrhage in internal organs, paralysis, respiratory failure, death
Roshchin[74]	Rat	Dust, fume	10 to 30 3 to 5	2 hr/day, for several months	Hemorrhagic inflammation in lungs, purulent bronchitis, pneumonia
Pazynich[75]	Rat	Fume	0.027	Continuous, 70 days	Hemorrhagic inflammation in lungs, vascular congestion and hemorrhage in liver, kidneys, and heart

not product acute toxicity, perhaps because particles were too large. Acute toxic effects, however, were observed following intratracheal instillation of ferrovanadium, which may be related to its biological solubility and degree of absorption.

Acute inhalation toxicity was characterized by irritation of the respiratory mucosa, with nasal discharge, sometimes containing blood. Animals breathed with difficulty and with crepitations. Behavior was passive; the animals refused to eat and lost weight. Dysentery, paralysis of the hind limbs, respiratory failure, and death ensued in cases of severe poisoning. Pulmonary abscesses were found frequently in animals that recovered. Animals that died or were killed at various times after exposure showed severe congestion, particularly in the capillaries, and tiny hemorrhages in all internal organs. There was evidence of increased intracranial pressure. Livers and kidneys showed fatty degeneration. Lungs showed capillary congestion, tiny hemorrhages, perivascular and focal edema, bronchitis, and focal interstitial pneumonia. The bronchitis and bronchopneumonia were often purulent, and the small bronchi were constricted. The severity of pathological changes could be related to vanadium content in the air, and in the cases of slight toxicity, the pathological changes were mainly observed in the lungs.

When rats were exposed intermittently to a condensation aerosol of vanadium pentoxide 2 hours every other day for 3 months at 3 to 5 mg/m^3 (or a grinding aerosol of V_2O_5 at 10 to 30 mg/m^3 for 4 months), pathological changes were seen only in the lungs. Blood vessels of the lungs were engorged with blood and showed a swollen endothelium; there were capillary congestion, perivascular edema, lymphostasis, and tiny hemorrhages indicating altered vascular permeability and disturbances of pulmonary blood and lymph circulation. Foci of edema were sometimes seen, and, in some cases, there was desquamative bronchitis. Small bronchi were often constricted. Interstitial tissue was infiltrated by lymphocytes and histiocytes. Connective tissue proliferation was sometimes seen in the zone of lymphocytic infiltration. Some animals showed purulent bronchitis or pneumonia, and occasionally lung abscesses developed.

Similar effects were observed with vanadium trioxide and vanadium trichloride;[31] the latter compound, being more soluble, showed more marked histopathological effects on internal organs. Pentavalent compounds of vanadium were three to five times more toxic than those of trivalent vanadium (in terms of median lethal concentration). Grinding aerosols of vanadium metal, vanadium carbide, and ferrovanadium were not highly toxic; however, chronic exposure to them at high concentrations produced many of the same symptoms as described above for vanadium pentoxide.

In summary, the basic manifestations of vanadium exposure of the experimental animals were, according to Roshchin:[74]

- Marked irritation of the respiratory mucosa.
- Vascular injury that resulted in capillary stasis, perivascular edema, and tiny hemorrhages, i.e., a hemorrhagic inflammation process.
- A spastic effect on the smooth muscle of the bronchi that resulted in asthmatic-type bronchitis and expiratory difficulty on acute exposure.
- Vascular changes — resulting from absorption — in internal organs and brain, which in turn cause neurological symptoms, toxic nephritis, and disorders of protein metabolism.

With respect to the respiratory tract in experimental animals, the major differences between acute and chronic effects of vanadium relate to the development, with prolonged exposure, of chronic inflammation in bronchi, accompanied by greater tendency to septic bronchopneumonia. Atelectasis, interstitial infiltration and proliferation, and emphysema were also noted.

The histopathological changes observed in kidney and liver following *acute* exposure to vanadium at high concentrations (tens of milligrams per cubic meter) are not usually seen with *intermittent* low-level exposure.

Other physiological effects have been mentioned in cases of severe exposure of animals to oxides and salts.[51] These include disturbances of the central nervous system (impaired conditioned reflexes and neuromuscular excitability) and cardiovascular changes (occurrence of arrhythmias and extrasystole, prolongation of the Q-RST interval, and decrease in the height of the P and T waves of the EKG). The significance of these findings with respect to human environmental exposure, if any, is not clearly defined at the present time.

6.1.5.3. Metabolic Effects — In the studies described above by Roshchin[74] and in his subsequent investigations,[18] a number of metabolic alterations were observed. After exposure of rabbits to a grinding aerosol of vanadium trioxide (40 to 75 mg/m^3, 2 hours/day for up to 12 months), several changes were reported: the test animals inhibited a progressive weight loss amounting to an average of 4.6 percent at the termination of the experiment; whereas controls gained weight by 12.3 percent. The number of blood leucocytes declined after the fifth month from between 7,000 and 9,000/mm^3 to 5,000/mm^3 by the end of the test; whereas no change was noted in controls. Hemoglobin levels in the test animals decreased from 75 to 68 percent. A normal rabbit hemoglobin is 8 to 15 g/100 ml, and a normal rabbit hematocrit is 30 to 50 percent. Serum ascorbate levels in the blood progressively decreased to about 20 percent of control between 7 and 8 months. Protein sulfhydryl levels in the serum of exposed animals decreased by 30 percent as compared with controls. Respiration in liver and brain tissue of test animals was reduced by one-half by the end of the experiment as compared with controls, but the respiratory quotient was unchanged. Blood cholinesterase in exposed rabbits increased by an average of 25 percent after the fifth month.

In these studies, the weight loss along with the depressions in the levels of white cells, hemoglobin, and protein sulfhydryl groups in the blood and the decreased tissue respiration were taken as indicators of the "general toxic effect" of vanadium. Increased cholinesterase activity was held to be indicative of sensitization. Bronchial asthma was taken as a clinical symptom of sensitization.

Chronic poisoning from the inhalation of trivalent vanadium (V_2O_3 and VCl_3) resulted in blood changes by the end of the second and third month. These changes were characterized by decreased albumin and increased globulins (mainly γ globulins) such that the albumin-globulin ratio was halved; and by an increase in serum concentrations of three amino acids—cystine, arginine, and histidine. There was also a 10 precent increase in nucleic acid in the blood and a "considerable" increase in blood chloride. Roshchin[51] has said that the "effect of vanadium on the metabolism of proteins and nucleic acids is responsible for the immunological and allergic reactions important manifestations of vanadium poisoning." This suggests that vanadium can act as a hapten in elicting an immunological response, but no supportive evidence is available.

In an attempt to explain the mechanism of the initial nonspecific hematopoetic effect of vanadium and the subsequent anemia, Roshchin[51] hypothesized that

> . . . the redox system of hydrogen carriers is inhibited or blocked, and in response to the resulting hypoxia, there is increased regeneration of the formed elements of the blood . . . Pos-

> sibly vanadium interferes with tissue respiration at the state of dehydrogenation effected by coenzyme I* belonging to the group of dehydrases [sic]. By inhibiting this coenzyme, vanadium (similarly to lead) interferes with the incorporation of iron in the porphyrin complexes, thereby retarding the synthesis of hemoglobin. The anti-vitamin C effect of vanadium is closely related to the inhibition of hemoglobin synthesis. Vitamin C deficiency in the body likewise inhibits the utilization of iron for hemoglobin synthesis, iron becoming accumulated in the reticuloendothelial tissue. . . .

Roshchin also points out that vanadium is known to inhibit the activity of monoamine oxidase, which catalyzes the conversion of serotonin to 5-hydroxyindoleacetic acid. In rabbits chronically exposed to vanadium pentoxide dust for 3 months, Roshchin observed that urine levels of 5-hydroxyindoleacetic acid had fallen to 33 percent below control values. He suggests, therefore, that inhibition of monoamine oxidase may result in accumulation of serotonin in the central nervous system, leading to functional disturbances. The sensitivity of smooth muscle to accumulation of serotonin, he notes, could result in bronchospasm, diarrhea, and urinary incontinence. The dystrophic and necrotic process in the kidneys and the high permeability of the blood vessels could, in his opinion, also be explained by elevated serotonin levels.

Roshchin also suggests an interaction of vanadium with an unspecified enzyme to account for the observed decrease in sulfhydryl groups in blood proteins and to explain the reduced cystine content of keratinized tissues.

Many of the clinical findings observed and interactions hypothesized by Roshchin and others can be accounted for or amplified by examination of the rather fragmentary knowledge of biochemical effects of vanadium exposure in experimental animals and *in vitro.*

Bergel et al.[76] reported that the catabolism of cystine and cysteine is increased by exposure to vanadium. In studies *in vitro,* Anbar and Inbar[77] demonstrated that, in the presence of VO^{2+}, pyridoxal 5-phosphate induces the catabolism of sulfhydryl amino acids. They pointed out that the activation of pyridoxal phosphate by vanadyl ions is rather specific to α-, β-elimination, and it strongly suggests a decrease of -SH groups in the organism.[77] This observation provides a corollary to the observed lowering of cystine levels in keratinized tissues.[64,65] Decreased synthesis of cysteine and cystine was thought to account for reduced levels of cystine in hair and nails. The essential point, however, is that metabolic processes that depend on either of these amino acids may be depressed in the presence of vanadium.

Cysteine is required in the biosynthesis of coenzyme A, being added to 4-phosphopantothenic acid in the presence of adenosine triphosphate (ATP) to form the intermediate 4'-phosphopantothenyl cystine. Coenzyme A plays a central role in many biosynthetic and oxidative pathways.[78] Mascitelli-Coriandoli and Citterio[79,80] have demonstrated that treatment with sodium metavanadate lowers the content of coenzyme A in rat liver. (The administration of an antimetabolite of pantothenic acid, -methylpantothenic acid, to man results in a syndrome consisting of postural hypotension, dizziness, tachycardia, fatigue, drowsiness, epigastric distress, anorexia, numbness and tingling of hands and feet, and hyperactive deep reflexes. It is not known whether these symptoms reflect an induced deficiency of pantothenic acid or the toxicity of the antimetabolite. The symptoms, however, are not unlike those resulting from exposure to high concentrations of vanadium.

---

*Coenzyme I was previously used to denote nicotinamide adenine dinucleotide (NAD).

The common denominator in both cases may be reduced hepatic coenzyme A levels.)

The requirement for coenzyme A in biochemical pathways where acetate is a starting material suggests that these processes will be impaired by excessive exposure to vanadium. In 1954, Curran[81] demonstrated that the synthesis of cholesterol from acetate-^{14}C in rat liver was diminished in the presence of vanadium. Subsequently, Azarnoff et al.[82,83] showed that one site of inhibitory action of vanadium in the cholesterol biosynthetic pathway was at the level of squalene synthetase — the enzyme that catalyzes the conversion of farnesyl pyrophosphate to squalene. Vanadium was also shown to mobilize aortic cholesterol in atherosclerotic rabbits more rapidly than was the case in controls.[84] With respect to the observed[81] effect of vanadium in lowering circulating cholesterol levels, Curran and Burch[15] recently suggested that a regulatory enzyme, acetoacetyl coenzyme A deacylase, for the biosynthesis of cholesterol, is activated by vanadium in young animals but inhibited by vanadium in older ones. This suggestion may explain the fact that cholesterol levels appear to be lowered by vanadium in younger animals, including humans, and not in older ones.

Because acetyl coenzyme A is a precursor of fatty acids, it has been suggested that vanadium may depress the synthesis of triglycerides and phospholipids. Levels of triglycerides were decreased in livers of rats given vanadium;[85] however, serum triglycerides in men were increased following ingestion of vanadium.[86] The incorporation of labeled phosphate into liver phospholipids was decreased following injection of vanadyl sulfate.[87] This finding could have resulted from inhibition of phospholipid biosynthesis or from increased oxidative degradation as suggested by the Bernheims.[88,89]

Coenzyme A is also required in the synthesis of coenzyme Q, or ubiquinone, of the mitochondrial electron transport chain. Aiyar and Sreenivasan[90] demonstrated that ubiquinone synthesis in isolated mitochondria was reduced in the presence of vanadium. When cysteine was given with vanadium, the effect on ubiquinone synthesis was partially reversed. Further addition of ATP and coenzyme A completely prevented the inhibition of ubiquinone synthesis.

Coenzyme A is required in the biosynthesis of many other biochemicals; however, the effect of vanadium on these biosynthetic processes has not been investigated.

Wright et al.[91] have demonstrated that vanadium uncouples mitochondrial oxidative phosphorylation in liver homogenates *in vitro,* resulting in depletion of the ATP energy stores. The addition of ammonium metavanadate to the diet at a level to supply 25 μg/g vanadium also uncoupled oxidative phosphorylation in liver mitochondria of young chicks.[92] The mechanism of uncoupling has not been delineated. Hathcock et al.[92] suggest that vanadate may replace the phosphate ion in the reactions leading to the synthesis of ATP such that a vanadyl intermediate or an ADP-V is formed.

Aiyar and Sreenivasan[90] have shown that vanadium salts inhibit succinic dehydrogenase, which would also reduce ATP synthesis. Succinic dehydrogenase, a key enzyme of the citric acid cycle and the electron transport system, is activated by sulfhydryl groups. Vanadium could inhibit this enzyme by mediating a decrease in available -SH groups. Sulfhydryl groups are also involved in the regulation of the deiodination of thyroxine at the cellular level.[77] Thyroxine, along with Ca^{2+} and perhaps other agents, acts through a so-called "U-factor" to cause swelling of mitochondria and uncoupling of oxidative phosphorylation.[78]

Perry et al.[93,94] have reported that, in the presence of vanadium, the oxidation of tryptamine by monoamine oxidase from guinea pig liver and kidney was accelerated by 125 percent. Studies by

Lewis,[95] however, indicated that vanadium inhibited monoamine oxidase, because the urinary output of 5-hydroxyindoleacetic acid was reduced in dogs injected with sodium metavanadate. Decreased output of 5-hydroxyindoleacetic acid suggests the possibility of accumulation of serotonin as previously found by Roshchin.[51]

Other health-related effects of vanadium have been noted in the literature. Vanadium has been reported to decrease the incidence of dental caries when added to the diet of hamsters.[96] Subsequent studies[97-101] have failed to demonstrate a clearly beneficial effect with regard to dental caries in humans, however. Vanadium in high concentration has been reported to reduce hemoglobin content and to produce anemia in experimental animals.[51] However, when low levels of vanadium were administered to men or animals with a normal hemoglobin level, little effect was observed.[72,102] Further reports suggest beneficial effects of low-level vanadium in treating nutritional anemia.[72,100,103,104]

In a study of goiter in two areas in the Kiev region of Russia, Barannik et al.[105] found food levels of vanadium and chromium to be higher in the goitrous area than in the comparison area. No other similar observations have been reported.

No data were found on carcinogenic, mutagenic, or teratogenic effects of vanadium exposure in humans or experimental animals.

6.1.5.4. Functional Effects — Two studies have reported changes in the functional state of mice and rats following exposure to vanadium. In a series of studies by Selyankina,[106] dissolved vanadium pentoxide or ammonium vanadate was administered orally to rats or mice in doses of 1 to 0.005 mg V/kg body weight/day for periods of from 21 days at the higher levels to 6 months at the lower levels. The threshold dose causing functional disturbances of conditioned reflex activities in mice and rats was 0.05 mg V/kg body weight. A dose of 0.005 mg V/kg body weight proved to be inactive. On the basis of these experiments, a vanadium concentration of 0.1 mg/liter was recommended as a maximum permissible concentration for water basins in Russia. This level was calculated to permit an intake of 0.005 mg V/kg body weight/day by a 60-kg man consuming 3 liters of water.

A report by Pazynich[75] involved continuous inhalation exposure of rats for 70 days to condensation aerosols of vanadium pentoxide at levels of 0.027 ± 0.002 mg/m^3 and 0.002 ± 0.00013 mg/m.^{3} Rats in both groups experienced normal gains in weight, as did the controls. After 30 days, in the rats exposed at the higher level (0.027 mg/m)^3, the motor chronaxy of the extensor muscles of the tibia decreased by an average of 0.8 microsecond (p. <0.01), and the chronaxy of the corresponding flexor muscles increased by an average of 4 microseconds (p. <0.001). Thus, the chronaxy ratio of antagonistic muscles had fallen from 1.5 at the beginning of the experiments to 1.0 on the 20th day (p. <0.02), to 0.5 on the 30th day (p. <0.01), and finally to a level of about 0.25. The chronaxy ratio returned to normal (1.5) on the 70th day, about 18 days following cessation of exposure. No changes were observed in motor chronaxy in controls or in rats exposed at the lower level (0.002 mg/m^3). Statistically significant changes in other parameters observed at the high level exposure (0.027 mg/m^3), but not at the lower level, included depressed whole blood cholinesterase, decreased total serum protein, depressed serum β-gobulins, and decreased oxyhemoglobin content of venous blood. Also observed in the high level exposure group were elevated serum γ-globulins; increased number of blood leucocytes showing yellow, orange, and red nuclear fluorescence with acridine orange; and increased oxygen consumption as indicated by the minced livers. The pattern of leucocyte nuclear fluorescence returned to normal 20 days following cessation of exposure. Histopathological changes observed following high level exposure included marked lung congestion, focal lung hemorrhages, and extensive bronchitis. Liver changes included central vein congestion with scattered small hemorrhages, scattered infiltration between lobes, and granular degeneration of hepatocytes. The kidneys showed marked granular degeneration of the epithelium of the convoluted tubules. In the heart, myocardial vascular congestion was observed with focal perivascular hemorrhages.

Because of the large differences in concentrations employed in the first two experimental groups (0.027 and 0.002 mg/m^3), a second experiment was performed in which rats were exposed continuously to vanadium pentoxide at 0.006 ± 0.00056 mg/m^3 for 40 days. During the first month of exposure, no changes as compared to controls were observed in the parameters investigated — that is, chronaxy of antagonistic muscles of the tibia and blood leukocyte nuclear fluorescence. After 30 days, there was a statistically significant decrease in chronaxy ratio. During the sixth week of exposure, animals were stressed by receiving only water and no food. After 3.5 days of this treatment, chronaxy ratios decreased to 0.92 as compared to 1.5 in controls, and the number of leukocytes displaying altered nuclear fluorescence increased by a factor of 4.83. The overall results of the study led Pazynich[75] to recommend the no-effect level of 0.002 mg/m^3 as the mean daily maximum permissible concentration of vanadium pentoxide in the atmosphere. Such a direct extrapolation of results from laboratory animal experiments to human community exposure is rarely, if ever, appropriate. This is particularly true for aerosol exposures of rodents in chambers because they constantly groom themselves with their tongues. They therefore obtain considerable doses of the aerosol by mouth in addition to the amount inhaled.

6.2. EFFECTS IN PLANTS AND MICROORGANISMS

The effects of vanadium on plant growth are variable. Cannon,[107] Allaway,[108] and Pratt[109] all mention that studies by a number of different researchers have shown that, in general, low levels of vanadium are beneficial to plant growth and that high levels are toxic. Vanadium has not been proved to be essential for higher-plant growth.[107,109]

Warington[110] studied the physiological effects of vanadium on plant growth. Apical iron-deficiency chlorosis followed a preliminary deepening of color in the shoot when plants were grown in nutrient solutions. Cannon[107] reports that reddening in plants similar to that mentioned by Warington had been observed around the uranium-vanadium deposits in Utah and Colorado. Geological Survey personnel[107] conducted experiments with sorghum and *Astragalus preussi* (a selenium-accumulating legume). The plants were grown in nutrient solutions containing 1, 10, and 100 $\mu gV/g$ as ammonium vanadate. No effect on growth of germinated seeds of sorghum was noted as 1 $\mu g/g$; at 10 $\mu g/g$, reddening first of the lower stems and later of the leaf tips was observed; at 100 $\mu g/g$, stunting and death occurred after 2 weeks. In the same experiment, *A. preussi* was not affected by 100 $\mu g/g$ and developed roots 50.8 cm long in 6 weeks.

A. preussi is an accumulator of vanadium as well as selenium. When growing in the same soil, *A. preussi* showed a vanadium content of 400 $\mu g/g$ and *A. pattersoni* had 30 $\mu g/g$. Cannon[107] has suggested that the difference in uptake is related to the high calcium levels in the latter species. *A. pattersoni* contains nearly twice as much calcium as *A. preussi*. Vanadium, it was suggested, is precipitated by calcium to calcium vanadate in the roots of *A. pattersoni*, thus reducing its mobility.

Absorption of vanadium from carnotite, $K_2(UO_2)_2(VO_4)_2 \cdot 3H_2O$, was compared with that from other types of deposits by analyzing the vegetation growing in the soil types mentioned.[107] The ratio of vanadium in the soil to vanadium in plant ash usually approximated 10 to 1. The absorption of vanadium by plants was low. From rocks that contain large amounts of calcium — for example, tyuyamunite, $Ca(UO_2)_2(VO_4)_2.nH_{2}0$ — and also from sandstones containing calcium. Plant absorption was high on sandstones and shales that supported seleniferous vegetation.

Plot experiments set up to determine the effects of carnotite ore and some of its toxic components (uranium, vanadium, and selenium) on plant life showed that plant growth was stimulated in the plots to which carnotite had been added.[107] The relationship between absorption of vanadium and pH was not consistent. In the plots in which $CaSO_4$, $CaCO_3$, and $Ca_3(PO_4)_2$ were present, the vanadium content in the above-ground plant parts was low; but in plots containing selenium, vanadium was high (Table 6.14). In calcium plots, the root-to-shoot ratio of vanadium was found to be greater (Table 6.15). In general, the presence of calcium in the soil seems to lessen the vanadium uptake by plants.[107]

Prince[111] attempted to correlate the vanadium content of the soil with the concentration found in corn plants grown in the soil. He found that the total supply of trace elements in the soil has a definite bearing on the amount found in the corn plant, but that factors that influence the availability of the elements (the pH, mineral form, and solubility) are more important in determining plant uptake.

Molybdenum plays an essential role in assimilatory nitrate reduction. The metal is essential for microbial growth with nitrate but not with ammonia.[112] Vanadium may replace molybdenum in the nitrogen-fixing process mentioned above among certain strains of *Azotobacter*, a free-living, nitrogen-fixing bacterium; [113,114] molybdenum, however, is more active. [115]

Cannon[107] suggests that vanadium may also stimulate nitrogen fixation by bacteria living in the root nodules of leguminous plants. Pratt[109] cites the work of Nicholas[116] who reported that vanadium played a role in nitrogen fixation by microorganisms living in root nodules. Pratt[109] also mentions studies in which the addition of ammonium metavanadate and calcium vanadate to soil resulted in increased growth in red clover. Studies using plants other than legumes showed little or no growth stimulation.[109] Bertrand[117] first noted that the root nodules of legumes contained higher concentrations of vanadium than the roots of other plants.

Cannon[107] reported that vanadium in concentrations of 10 to 20 $\mu g/g$ in solution was toxic to plants. Pratt,[109] citing various studies, noted that vanadium added to nutrient solutions in concentrations of 0.5 $\mu g/g$ or greater is toxic to plants. Toxicity in a variety of crops has been produced by adding vanadium to the soil. Toxicity symptoms were extreme dwarfing and chlorosis.

Vanadium as ammonium vanadate was found to be essential for the growth of the green alga, *Scenedesmus obliquus*. The presence of vanadium in the medium stimulated photosynthesis and growth of the alga. It could not be replaced by molybdenum or other metals.[118]

Microbial action plays a strong role in determining the availability of vanadium in the soil. The bi-, tri-, and pentavalent states of vanadium make it susceptible to microbial attack. Specific autotrophic bacteria oxidize the reduced forms of vanadium to obtain part or all of their energy for growth multiplication.[115] *Micrococcus lactilyticus* in the presence of hydrogen is able to reduce vanadate,

Table 6.14. VANADIUM IN ASH OF ABOVE-GROUND PARTS OF PLANTS GROWN IN EXPERIMENTAL PLOTS[104] ($\mu g/g$)

Plant and year	Vanadium in plant ash[a]					
	Control (soil pH 6.6)	Carnotite $[K_2(UO_2)_2(VO_4)_2.3H_2O]$ (soil pH 7.3)	Carnotite + $CaSO_4.H_2O$ (soil pH 7.0)	Carnotite + $CaCO_3$ (soil pH 6.9)	Carnotite + $Ca_3(PO_4)_2$ (soil pH 6.5)	Carnotite + Na_2SeO_3 (Soil pH 7.7)
Grindelia aphanactis, 1954	50	105	25	15	25	84
Inscurainia obtusa, 1951	<15	100	24	—	20	250
Eschscholtzia californica, 1954	<15	<15	30	<15	<15	75
Verbesina enccliodes, 1954	10	25	<15	<15	10	20

[a] Soils of each plot contained $<6 \mu g/g$ water-soluble vanadium.

Table 6.15. COMPARISON OF VANADIUM IN ASH OF TOPS AND ROOTS OF PLANTS GROWN IN EXPERIMENTAL PLOTS[104]

Plant and year	Plot	pH	Vanadium concn., $\mu g/g$		
			Tops	Roots	Roots/tops
Verbesina, 1955	Carnotite plus calcium phosphate	7.5	10	1,500	150
Stanleya, 1957	Calcium carbonate	7.7	<10	70	>7
Grindelia, 1955	Carnotite plus gypsum	7.7	35	500	14
Grindelia, 1956	Carnotite	6.7	50	150	3
Astragalus preussi, 1956	Sodium selenite	7.9	50	150	3

molybdate, selenite, tellurite, and arsenate[115] (Figure 5.8). Iron-oxidizing bacteria, such as *Thiobacillus ferrooxidans* and *Ferrobacillus thiooxidans*, oxidize vanadium and make it more soluble in aqueous solutions and, therefore, capable of transport in groundwater and of plant uptake. Sulfate-reducing bacteria form hydrogen sulfide and provide reducing conditions for the reduction of pentavalent vanadium and the formation of vanadium sulfides. The reduction of vanadium sulfide to trivalent vanadium is catalyzed by *Micrococcus lactilyticus* under the above conditions.[115]

The mushroom, *Amanita muscaria*, is an accumulator of vanadium. Neither the significance of nor the reasons for this accumulation are known;[117] however, vanadium has been shown to be growth-stimulating to amanitas. Bertrand[117] noted that vanadium is not localized in any one part of the mushroom. When eaten in small quantities, the mushroom is hallucinogenic; in large amounts, it is poisonous. Much has been written about the properties of this mushroom and its use in orgiastic festivals by Siberian tribes.[119] The hallucinogenic properties have been associated with muscarin, an alkaloid found in the mushroom. Muscarin does not have vanadium as a component.[120]

Vanadium toxicity in plants is not usually found in agricultural conditions.[118] The primary consideration is toxicity to humans and other animals through food ingestion.

6.3. EFFECTS IN ANIMALS

Vanadium has been found in the tissues of representative species from all animal phyla,[107] and the concentrations have been determined.[7,117] Most studies dealing with physiology, metabolism, and toxicity have used experimental animals.

Ascidians (sea squirts), which belong to the Notochordata, have high concentrations of vanadium in their blood. The serum concentration often reaches 10,000 times that of sea water.[7] The function of chromogen (trivalent vanadium complexed to pyrrole rings), in vanadocytes (green blood cells containing hemovanadin or chromogen), is not known. It was once thought that chromogen was an oxygen carrier in the blood, but it has since been shown that it acts as a reducing agent.[107]

Rock phosphates and colloidal clays are sometimes used as phosphate supplements for growing chicks.[121] These phosphates have been found to be high in vanadium and chromium. Experiments showed that vanadium was the more significant of the two in its influence on chick growth. A concentration of 30 $\mu g/g$ of vanadium as calcium salt depressed growth; over 200 $\mu g/g$ resulted in mortality. Chromium at levels up to 100 $\mu g/g$ caused no effect.[121]

Three commercial samples of tricalcium phosphate, two of dicalcium phosphate and one of bone meal, were compared in chick diets by Berg.[122] A significant depression in growth was associated with the two samples of tricalcium phosphate. The growth depression was associated with the vanadium (ammonium metavanadate) content of the tricalcium phosphate.

Hathcock et al.[123] showed that levels as low as 25 $\mu g/g$ of vanadium, as either ammonium metavanadate or as vanadyl sulfate, were toxic to chicks. Disodium ethylenediaminetetraacetate, when added to the diet, prevented vanadium toxicity.

The possibility that trace elements could be toxic when ingested at low levels over long periods of time was studied by Schroeder and Balassa.[124] Rats and mice showed no effects when fed vanadyl sulfate over a lifetime, but vanadium did accumulate in some of the organs.[124] The fact that vanadium has been determined essential for growth in rats[125] may explain the absence of toxicity when it is fed to them.

Ter Heege[126] reported an incident in which four milk cows were exposed to soot cleaned from an oil-heated boiler. The soot, dumped near a pasture, was spread by the wind to the extent that the pasture grass was covered with a film of soot. Of the animals exposed, one died, one became very ill, and one showed no symptoms of illness. The quantity of vanadium consumed is not known. The soot contained 1 μg V/g. The animal that became very ill was slaughtered. Toxicological examination showed 1.5 μg V/g liver and 3 μg V/g kidney (both wet weight) and 2.85 mg V/liter of urine. Analysis on one of the animals that died of poisoning showed 2.4 μg V/g in liver, 4.3 μg V/g in renal cortex, and 4.7 μg V/g in renal marrow. Pathological evidence obtained by autopsy led to speculation that vanadium was the cause of illness and death in this incident.

6.4. EFFECTS ON MATERIALS AND THE ENVIRONMENT

6.4.1. Metal Corrosion

Vanadium dispersed in air, water, or soil has not been reported to produce or promote damage to metals or other materials. High concentrations may cause damage in certain situations; however, the most important and troublesome situation is caused by ash deposits in oil-fired heating and power generating units that burn residual or crude oils. The problem is particularly acute with the use of high-vanadium oils. The ash deposits remain on the metal surfaces for long periods and are difficult to remove. Much of the vanadium in the fuels remains in this ash. Earner in this chapter, the industrial hygiene aspects of vanadium exposure are discussed. Removal of this ash is a major source of injury from occupational exposure to vanadium. Major damage to boilers, pipes, and other metal objects in these plants is also caused by vanadium in the ash. Direct contact with vanadium corrodes the metals. Vanadium also catalyzes the oxidation of SO_2 formed from sulfur in the fuel to SO_3, with subsequent formation of highly corrosive sulfuric acid. The usual methods of dealing with these problems are cleaning periodically and using oils with low levels of vanadium and sulfur.

During the last decade, efforts to find other means of dealing with the corrosion problem have included the use of fuel additives. Metallic additives, particularly magnesium oxides and methyl cyclopentadienyl manganese tricarbonyl, have been reported to be successful. The overall problem and the attempted solutions were described in an unsigned paper in *Modern Power and Engineering* in 1971.[127] Exley et al.[128] described the use of magnesium oxide at the Long Island Lighting Company. This fuel treatment, combined with low excess air, appears to have made considerable improvement in plant operation. The resultant ash, which can be sold for vanadium recovery, is less corrosive and more easily removed. Use of manganese as an additive is described by Belyea[129] and by Papamarcos.[130] Low excess air is not used in this operation, but the effects appear to be somewhat similar. The mechanisms involved in these empirical observations are not entirely understood.[131] Effects on emissions of vanadium, sulfur compounds, magnesium, manganese, etc., are not known, but it appears that some alterations would be present.

6.4.2. Vanadium in Sludges and Other Wastes

Some information is available on concentrations of vanadium in waste materials. Because these wastes are disposed of in landfills, in the sea, or by incineration, some knowledge of the quantities of metals involved and their distribution is of interest and value. Occasionally, as with "oil ash," possibilities of valuable resource recovery are identified. Thompson et al.[132] studied the metal content of sewage sludge from 12 urban plants in Oklahoma. Analyses were made by emission spectrography, and the results are given in percentage of air-dried sludge. Vanadium levels ranged from 0.03 to 1.50 percent. One plant had 1.50 percent, two had 1.00 percent, and the remainder had 0.10 percent or less. The higher levels were thought to be related to higher concentrations in the local water supplies. Berrow and Webber[133] performed similar studies on 42 sewage sludge samples from a variety of urban and nonurban locations in England and Wales. Measurements were compared with those of typical uncontaminated British soils. Vanadium concentrations in sludge samples ranged from 20 to 400 $\mu g/g$, with a mean of 75 $\mu g/g$ and a median of 60 $\mu g/g$. Soil levels, by comparison, were 20 to 500 $\mu g/g$, with a mean of 100 $\mu g/g$. Concentrations of 0.88 to 7.5 $\mu g/g$ with a mean of 3.0 $\mu g/g$ vanadium were extractable using a method employing 2.5 percent acetic acid. This is about 4.8 percent of the total vanadium present in the sludge. Soils gave <0.05 to 1.0 $\mu g/g$ extractable vanadium, with a mean of 0.5 $\mu g/g$. Thus, fertilization with such sludges would not increase soil vanadium content, but might increase the amount of soluble metal.

Gross [134] has studied the composition of sewage sludges, coal ash, and other wastes that are disposed of in New York harbor. Figure 6.1[134,135] compares the levels in these wastes and in existing harbor sediments moved during channel dredging, and in some characteristic soils and rocks. Here also the concentrations in waste materials are close to those that occur naturally. The coal ash content is at the high end of the range, however.

6.4.3. General Environmental Contamination

Under certain circumstances, information can be obtained on environmental contamination over time — sometimes over periods of several centuries. Suitable material for such studies is found in old botanical collections, old bones, old wood and tree rings, ice and snow from glaciers and polar areas, etc. Although it is rarely looked for in these studies, vanadium is included in a current study of glacier ice by Jaworowski et al.[136] Figure 6.2 is a graph from the first part of this study. Vanadium levels are low, but they show a small increase in the most recent decade. Lead and cadmium levels, in comparison, increased much earlier in relation to the development of industry in the 18th and 19th centuries.

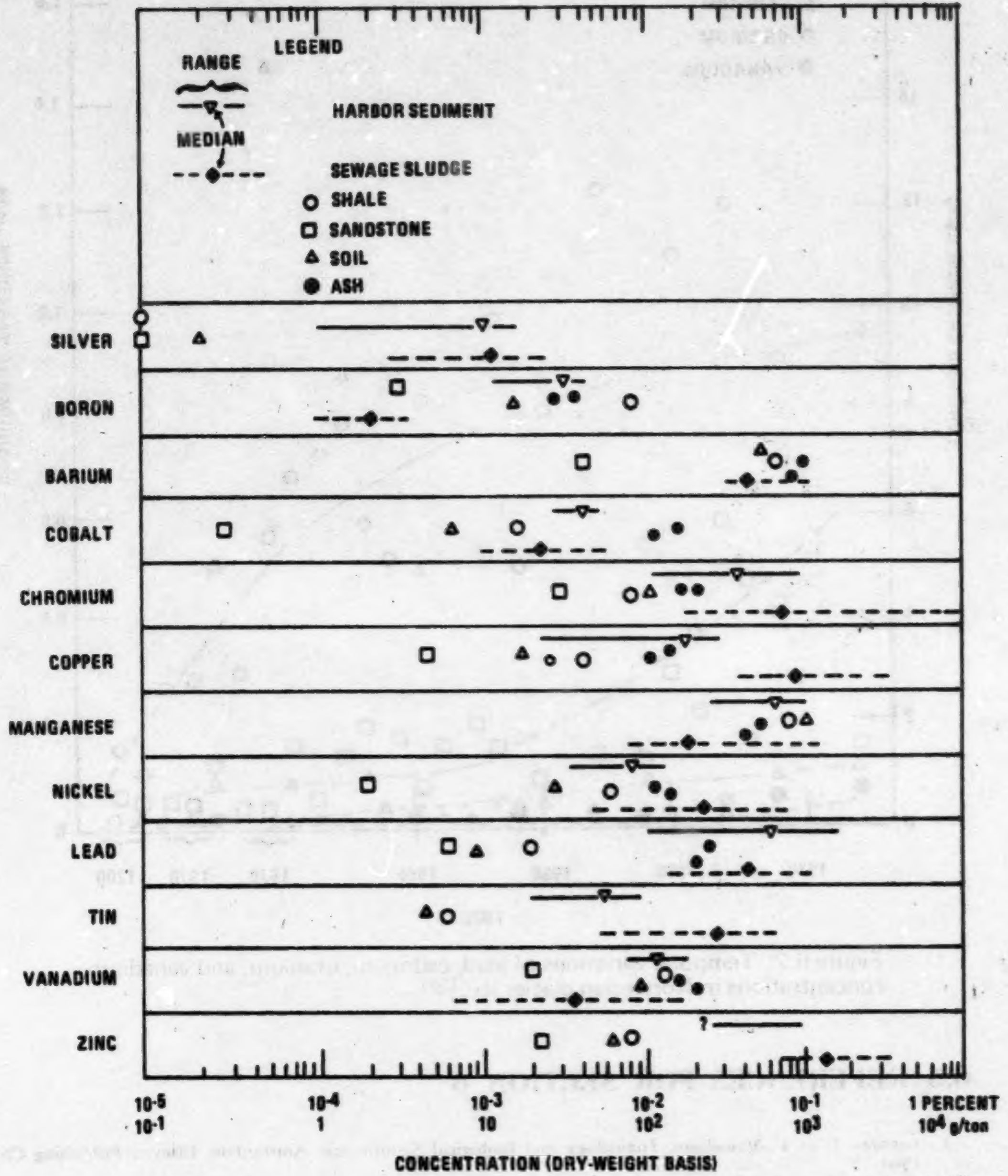


Figure 6.1. Chemical composition (minor elements) of harbor sediment and fly ash as compared with sewage sludges and various sediments and soils.[134,135]

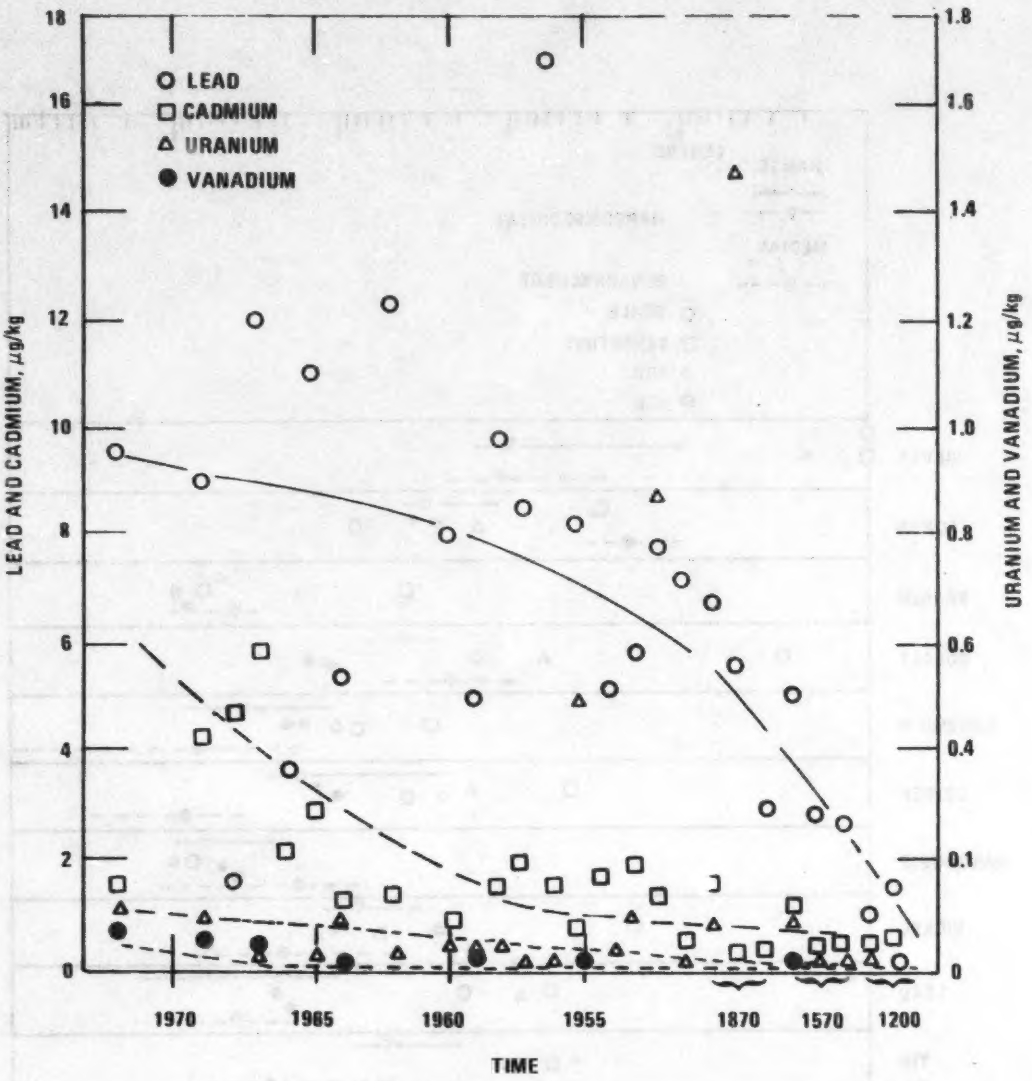


Figure 6.2. Temporal variations of lead, cadmium, uranium, and vanadium concentrations in Norwegian glacier ice.136

6.5 REFERENCES FOR SECTION 6

1. Hudson, T. G. F. Vanadium, Toxicology and Biological Significance. Amsterdam, Elsevier Publishing Co., 1964.

2. Schroeder, H. S., J. J. Balassa, and I. H. Tipton. Abnormal Trace Metals in Man — Vanadium. J. Chronic Dis. *16*:1047-1071, 1963.

3. Symanski, J. Gewerbliche Vanadinschadigungen, Ihre Enstehung und Symptomatologie (Industrial Vanadium Poisoning. Its Origins and Symptomatology). Arch. Gewerbepath. Gewerbehyg. (Berlin) 9:295-313, 1939.

4. Sjoberg, S. G. Vanadium Pentoxide Dust; A Clinical and Experimental Investigation on its Effect after Inhalation. Acta Med. Scand. *138*:1-188 (Suppl. 238), 1950.

5. Stokinger, H. E. Vanadium V. *In:* Industrial Hygiene and Toxicology. 2nd Ed., Vol. II. F. A. Patty (ed.). New York, Interscience Publishers, 1967. p. 1171-1182.

6. Athanassiadis, Y. C. Preliminary Air Pollution Survey of Vanadium and Its Compounds; A Literature Review. U. S. Department of Health, Education, and Welfare, Public Health Service, National Air Pollution Control Administration, Raleigh, N. C. NAPCA Publication No. APTD 69-48, October 1969. 91 p.

7. Schroeder, H. A. Air Quality Monographs. Vanadium. Vanadium Monograph No. 70-13. American Petroleum Institute, Washington, D. C., 1970. 32 p.

8. Tipton, I. H., P. L. Stewart, and J. Dickson. Patterns of Elemental Excretion in Long-Term Balance Studies. Health Phys. *16*:455. 1969.

9. Schroeder, H. A. A Sensible Look at Air Pollution by Metals. Arch. Environ. Health. *21*:798-806, December 1970.

10. Curran, G. L., D. L. Azarnoff, and R. E. Bolinger. Effect of Cholesterol Synthesis Inhibition in Normocholesteremic Young Men. J. Clin. Invest. *38*:1251-1261, 1959.

11. Sommerville, J. and B. Davies. Effect of Vanadium on Serum Cholesterol. Amer. Heart J. *54*:54-56, July 1962.

12. Lewis, C. E. The Biological Actions of Vanadium. II. The Signs and Symptoms of Occupational Vanadium Exposure. A.M.A. Arch. Ind. Health. *19*:497-503, May 1959.

13. Levina, E. N. Relationships between Solubility of Metal Compounds and Their Toxicity, Distribution and Elimination From the Body. Gig. Tr. Prof. Zabol. (Moscow) *16*:40-43, 1972.

14. Tipton, I. H., and J. J. Shafer. Statistical Analysis of Lung Trace Element Levels. Arch. Environ. Health. *8*:58-67, January 1964.

15. Curran, G. L., and R. E. Burch. Biological and Health Effects of Vanadium. *In:* Proceedings, First Annual Conference on Trace Substances in Environmental Health. U. of Missouri, Columbia, Mo., July 10-11, 1967.

16. Troppens, H. The Uncertainties of Vanadium Pentoxide Intoxication. Deutsch. Gesundh. (Berlin) *24*:1089-1092, June 5, 1969.

17. Hopkins, L. L. and B. E. Tilton. Metabolism of Trace Amounts of Vanadium-48 in Rat Organs and Liver Subcellular Particles. Amer. J. Physiol. *211*:169-172, 1967.

18. Roshchin, I. V., A. V. Il'nitskaya, L. A. Lutsenko, and L. A. Zhidkova. Effects on Organisms of Vanadium Trioxide. Fed. Proc. Trans. Suppl. *24*:611-613, July-August 1965.

19. Söremark, R., S. Ullberg, and L. E. Applegren. Autoradiographic Localization of V-48-Labelled Vanadium Pentoxide in Developing Teeth and Bones of Rats. Acta. Odont. Scand. *20*:225-232, 1962.

20. Söremark, R. and S. Ullberg. Distribution and Kinetics of $^{48}V_2O_5$in Mice. *In:* Use of Radioisotopes in Animal Biology and the Medical Sciences (Vol. 2). New York, Academic Press, 1962. p. 103-114.

21. Strain, W. H., W. P. Berliner, C. A. Lankau, R. K. McEvoy, W. J. Pories, and R. H. Greenlaw. Retention of Radioisotopes by Hair, Bone and Vascular Tissue. J. Nucl. Med. *5*:664-674, September 1964.

22. Söremark, R. Vanadium in Some Biological Specimens. J. Nutr. *92*:183-190, 1967.

23. Hopkins, L. L., and H. E. Mohr. The Biological Essentiality of Vanadium. *In:* New Trace Elements in Nutrition. Mertz, W., and W. E. Cornatzer (ed.). New York, Marcel Dekker, 1971.

24. Strasia, C. A. Vanadium: Essentiality and Toxicity in the Laboratory Rat. Thesis, Purdue University. Dissertation Abstr. *32*:646-B, 1971.

25. Schwarz, K., and D. B. Milne. Growth Effects of Vanadium in the Rat. Science. *174*:426-428, October 22, 1971.

26. Dimond, E. G., J. Caravaca, and A. Benchimol. Vanadium, Excretion, Toxicity, Lipid Effect in Man. Amer. J. Clin. Nutr. *12*:49-53, January 1963.

27. Jaraczewska, W., and M. Jakubowski. An Attempt of Evaluation of Vanadium Exposure in the Chemical Industry. Med. Practice *15*(6):375-383, 1964.

28. Dutton, W. F. Vanadiumism. J. Amer. Med. Ass. *56*:1648, 1911.

29. Rundberg, G. Moderna Yrkessjukdomsproblem i Sverige (Occupational Diseases in Sweden). Nord. Med. (Stockholm) *2*:1845, 1939.

30. Balestra, G., and F. Molfino. Alterazioni Pulmonari da Polveri Negli Operai Adetti All'estrazione del Vanadio Dalle Ceneri di Nafta (Changes Provoked by Dust in the Lungs of Laborers Occupied in Working Up Petroleum Ash Containing Vanadium). Rass. Med. Industr. (Turin) *13*:5, 1942. Abstract in J. Ind. Hyg. *26*:7, 1944.

31. Wyers, H. Some Toxic Effects of Vanadium Pentoxide. Brit. J. Ind. Med. *3*:177-182, 1946.

32. Wyers, H. Some Recent Observations on the Hazards in the Chemical Industry. *In*: Proceedings, Ninth International Congress of Industrial Medicine. Bristol, John Wright, 1948. p. 900.

33. Sjøberg, S. G. Possible Importance of Allergy in Diseases Caused by Inhalation of Inorganic Dust. Acta. Allerg. (Kbh). *4*:357, 1951.

34. Sjøberg, S. G., and K. G. Ringer. Skin, Eye and Respiratory Tract Symptoms Associated with Cleaning of Oil-fired Boilers: An Investigation with Special Reference to Vanadium in Blood and Urine. Nord. Hyg. Tidskr. (Stockholm) *37*(9-10):217-228, 1956.

35. Zenz, C., J. P. Bartlett, and W. H. Thiede. Acute Vanadium Pentoxide Intoxication. Arch. Environ. Health. *5*:542-546, 1962.

36. Frost, J. Vanadiumforgiftning ved Rensing af Oliefyrede Kedler (Vanadium Poisoning from Cleaning Oil-Fired Furances) Ugeskr. Laeg. (Copenhagen) *113*:1309, 1951.

37. Williams, N. Vanadium Poisoning from Cleaning Oil-fired Boilers. Brit. J. Ind. Med. *9*:50, 1952.

38. Fallentin, B., and J. Frost. Sundhedsfaren ved Rensing af Diefyrede Kedler: Stammende fra Vanadium og Svovlsyre i Soden (Illnesses from the Cleaning of Oil-Fired Furnaces: Originating from Vanadium and Sulfuric Acid in the Soot). (With summary in English.) Nord. Hyg. Tidskr. (Stockholm) *3*:58, 1954.

39. Sjøberg, S. G. Vanadium Bronchitis from Cleaning Oil-Fired Boilers. Arch. Ind. Health. *11*:505, 1955.

40. Thomas, D. L. G., and K. Stiebris. Vanadium Poisoning in Industry. Med. J. Aust., *1*:607, 1956.

41. Hickling, S. Vanadium Poisoning. N. Z. Med. J. *57*:607, 1958.

42. Roshchin, I. V. The Hazards of Occupational Vanadium Intoxications in Boiler Cleaning Operations at Electric Power Stations, Operating on Fuel Oil, and Prevention Problems. Gig. Truda. Prof. Zabol. (Moscow) *6*:17-22, 1962.

43. Pielsticker, F. Gesundheitsschadigungen durch Vanadiumverbindungen: Ihre Symtomatologie und Prognose (Damage to Health By Vanadium Compounds; Their Symptoms and Prognosis). Arch. Gewerbepath. Gewerbhyg. (Berlin) *13*:73, 1954.

44. Gulko, A. G. Zur Charakteristik des Vanadiums als Industriegift (The Characteristics of Vanadium as an Industrial Poison). Gig. Sanit. (Moscow) *21*:24, 1956.

45. Matantseva, E. I. Condition of the Respiratory Organs in Workmen Handling Vanadium Pentoxide. Gig. Truda. Prof. Zabol. (Moscow), 7:41, 1960.

46. Vintinner, F. J., R. Vallenas, C. E. Carlin, R. Weiss, C. Macher, and R. Ochoa. Study of the Health of Workers Employed in Mining and Processing of Vanadium Ore. Arch. Ind. Health. *12*:635, 1955.

47. Rajner. V. Effect of Vanadium on the Respiratory Tract. Cesk. Otolaryng. *9*:202-204, August 1960.

48. Roshchin, I. V. Vanadium Metallurgy in the Light of Industrial Hygiene and Issues of Prevention of Occupational Diseases and Poisoning. Gig. Truda. Prof. Zabol. (Moscow) *9*:3-10, September 1964.

49. Fear. E. C. and F. H. Tyrer. A Study of Vanadium Poisoning in Gas Workers. Trans. Ass. Ind. Med. Officers. *8*:153, January 1958.

50. Browne. R. C. Vanadium Poisoning from Gas Turbines. Brit. J. Ind. Med., *12*:57, January 1955.

51. Roshchin, I. V. Toxicology of Vanadium Compounds Used in Modern Industry. Gig. Sanit. (Moscow) *32*:26-32. 1967.

52. Shumann-Vogt. B. Health Hazards in Industry Caused by Vanadium. Zbl. Arbeitsmed. (Darmstadt) *19*:33-39, February 1969.

53. Waters. M. D., D. E. Gardner, and D. L. Coffin. Cytotoxic Effects of Vanadium on Rabbit Alveolar Macrophages *in Vitro*. Toxicol. Appl. Pharmacol. *28*(2):253-263, May 1974.

54. Waters, M. D., D. E. Gardner, C. Aranyi, and D. L. Coffin. Metal Toxicity for Rabbit Alveolar Macrophages *in Vitro*. Environ. Res. *9*:32-47, 1975

55. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment. American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio, 1961.

56. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1972. American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio, 1972.

57. Documentation of the Threshold Limit Values for Substances in Workroom Air (3rd ed.). American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio, 1971. 286 p.

58. McTurk, L. C., C. H. W. Huis, and R. E. Eckardt. Health Hazards of Vanadium Containing Residual Oil Ash. Ind. Med. Surg. *25*:29, 1956.

59. Tebrock, H. E., and W. Machle. Exposure to Europium-activated Yttrium Orthovanadate: A Cathodoluminescent Phosphor. J. Occupational Med. *10*:692-696, 1968.

60. Roshchin, I. V. Hygienic Aspects of Industrial Vanadium Aerosol. Gig. Sanit. (Moscow) *11*:49-53, 1952.

61. Stokinger, H. E. Unpublished Laboratory Data. U. S. Department of Health, Education, and Welfare, National Institute of Occupational Safety and Health, Cincinnati, Ohio, August 1973.

62. Zenz, C., and B. A. Berg. Human Responses to Controlled Vanadium Pentoxide Exposure. Arch. Environ. Health. *14*:709-712, May 1967.

63. Watanabe, H., H. Murayama, and S. Yamaoka. Some Clinical Findings on Vanadium Workers. Jap. J. Ind. Health. *8*(7):23-27, 1966.

64. Mountain, J. T., F. R. Stockell, Jr., and H. E. Stokinger. Studies in Vanadium Toxicology. III. Fingernail Cystine as an Early Indicator of Metabolic Changes in Vanadium Workers. A.M.A. Arch. Ind. Health. *12*:494-502, 1955.

65. Mountain, J. T., L. L. Delker, and H. E. Stokinger. Studies in Vanadium Toxicology; Reduction in the Cystine Content of Rat Hair. A.M.A. Arch. Ind. Hyg. Occup. Med. *8*:406-411, 1953.

66. Mountain, J. T. Detecting Hypersusceptibility to Toxic Substances. Arch. Environ. Health. *6*:357, 1963.

67. R. G. Keenan. Unpublished data. Cited in: Mountain, J. T. Detecting Hypersusceptibility to Toxic Substances. Arch. Environ. Health. *6*:357, 1963.

68. deJorge, F. B., L. C. G. Ferreira, and C. P. Tilbery. Vanadium in Charcot-Marie Muscular Atrophy. Rev. Bras. Med. *27*:303-307, June 1970.

69. Stocks, P. On the Relations between Atmospheric Pollution in Urban and Rural Localities and Mortality from Cancer, Bronchitis, and Pneumonia, with Particular Reference to 3, 4-Benzopyrene, Beryllium, Molybdenum, Vanadium and Arsenic. Brit. J. Cancer. *14*:397-418, September 1960.

70. Hickey, R. J., E. P. Schoff, and R. C. Clelland. Relationship Between Air Pollution and Certain Chronic Disease Death Rates; Multivariate Statistical Studies. Arch. Environ. Health. *15*:728-738, December 1967.

71. Pinkerton, C., D. I. Hammer, K. McClain, M. E. Williams, K. Bridbord, and W. B. Riggins. Relationship of Manganese and Vanadium in the Ambient Air to Heart Disease and Influenza-Pneumonia Mortality Rates. Unpublished staff study. U. S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N. C., June 1972.

72. Lewis, C. E. The Biological Actions of Vanadium. I. Effects Upon Serum Cholesterol Levels in Man. A.M.A. Arch. Ind. Health. *19*:419-425, 1959.

73. Browning, E. Toxicity of Industrial Metals. London, Butterworth's, 1962.

74. Roshchin, I. V. Vanadium. *In:* Toxicology of the Rare Metals (Izraelson, Z.I., ed.). AEC-tr-6710, 1967. p. 52-60.

75. Pazynich, V. M. Maximum Permissible Concentration of Vanadium Pentoxide in the Atmosphere. Gig. Sanit. (Moscow) English Ed. *31*(7-9):6-12, July-Sept. 1966.

76. Bergel, F., R. C. Bray, and K. R. Harrap. A Model System for Cysteine Desulphydrase Action: Pyridoxal Phosphate-vanadium. Nature. *181*:1654-1655, 1958.

77. Anbar, M., and M. Inbar. Effect of Pyridoxal 5-Phosphate in the Presence of Vanadyl Ions on the Deiodination of Thyroxine. Nature. *196*:1213, December 22, 1962.

78. Mahler, H. R., and E. H. Cordes. Biological Chemistry. New York, Harper and Row, 1966.

79. Mascitelli-Coriandoli, E., and C. Citterio. Effects of Vanadium Upon Liver Coenzyme A in Rats. Nature. *183*:1527-1528, 1959.

80. Mascitelli-Coriandoli, E., and C. Citterio. Intracellular Thioctic Acid and Coenzyme A Following Vanadium Treatment. Nature. *184*:1641, 1959.

81. Curran, G. L. Effect of Certain Transition Group Elements on Hepatic Synthesis of Cholesterol in the Rat. J. Biol. Chem. *210*:765-770, 1954.

82. Azarnoff, D. L., F. E. Brock, and G. L. Curran. A Specific Site of Vanadium Inhibition of Cholesterol Biosynthesis. Biochem. Biophys. Acta. *51*:397-398, August 5, 1961.

83. Azarnoff, D. L., and G. L. Curran. Site of Vanadium Inhibition of Cholesterol Biosynthesis. J. Amer. Chem. Soc. 79:2968-2969, 1957.

84. Curran, G. L. and R. L. Costello. Reduction of Excess Cholesterol in the Rabbit Aorta by Inhibition of Endogenous Cholesterol Synthesis. J. Exp. Med. *103*:49-56, 1956.

85. Curran, G. L. Effect of Certain Transition Group Elements on Hepatic Synthesis of Cholestrol in the Rat. J. Biol. Chem. *210*:765-770, 1954.

86. Curran, G. L., D. L. Azarnoff, and R. E. Bolinger. Effect of Cholesterol Synthesis Inhibition in Normocholesteremic Young Men. J. Clin. Invest. *38*:1251-1261, 1959.

87. Snyder, F., and W. E. Cornatzer. Vanadium Inhibition of Phospholipid Synthesis and Sulphydryl Activity in Rat Liver. Nature. *182*:462, 1958.

88. Bernheim, F., and M. L. C. Bernheim. Action of Vanadium on Tissue Oxidations. Science. *88*:481-482, 1938.

89. Bernheim, F., and M. L. C. Bernheim. The Action of Vanadium on the Oxidation of Phospholipids by Certain Tissues. J. Biol. Chem. *127*:353-360, 1939.

90. Aiyar, A. S., and A. Sreenivasan. Effect of Vanadium Administration on Coenzyme Q Metabolism in Rats. Proc. Soc. Exp. Biol. Med. *107*:914-916, Aug.-Sept. 1961.

91. Wright, L. D., L .F. Li, and R. Trager. The Site of Vanadyl Inhibition of Cholesterol Biosynthesis in Liver Homogenates. Biochem. Biophys. Res. Commun. *3*:264-267, 1960.

92. Hathcock, J. N., C. H. Hill, and S. B. Tove. Uncoupling of Oxidative Phosphorylation by Vanadate. Can. J. Biochem. *44*:983-988, July 1966.

93. Perry, H. M., Jr., S. Teitlebaum, and P. L. Schwartz. Effect of Antihypertensive Agents on Amino Acid Decarboxylation and Amine Oxidation. Fed. Proc. *14*:113-114, 1955.

94. Perry, H. M., P. L. Schwartz, and B. M. Sahagian. Effect of Transition Metals and of Metal Binding Antihypertensive Agents on Tryptamine Oxidase and Dopa Decarboxylase. Proc. Soc. Exp. Biol. Med. *130*:273-277, 1969.

95. Lewis, C. E. The Biological Actions of Vanadium. III. The Effect of Vanadium on the Excretion of 5-Hydroxyindolacetic Acid and Amino Acids and the Electrocardiogram of the Dog. A.M.A. Arch. Ind. Health. *20*:455-466, 1959.

96. Geyer, C. F. Vanadium. A Caries Inhibiting Trace Element in the Syrian Hamster. J. Dent. Res. *35*:590-595, 1953.

97. Hein, J. W. and J. Wisotzky. The Effect of a 10 ppm Vanadium Drinking Solution on Dental Caries in Male and Female Syrian Hamsters. J. Dent. Res. *34*:756, 1955.

98. Muhler, J. C. The Effect of Vanadium Pentoxide, Fluorides, and Tin Compounds on the Dental Experience in Rats. J. Dent. Res. *36*:787-794, 1957.

99. McLundie, A. C., J. B. Shepherd, and D. R. A. Mobbs. Studies on the Effects of Various Ions on Enamel Solubility. Arch. Oral Biol. *13*:1321-1330, 1968.

100. Hadjimarkos, D. M. Vanadium and Dental Caries. Nature. *209*:1137, March 12, 1966.

101. Hadjimarkos, D. M. Effects of Trace Elements on Dental Caries. Advan. Oral Biol. *3*:253-292, 1968.

102. Franke, K. W., and A. L. Moxon. The Toxicity of Orally Ingested Arsenic, Selenium, Tellurium, Vanadium and Molybdenum. J. Pharmacol, Exp. Ther. *61*:89-102, 137.

103. Myers, V. C. and H. H. Beard. Studies in the Nutritional Anemias of Rats. II. Influence of Iron Plus Supplements of Other Inorganic Elements Upon Blood Regeneration. J. Biol. Chem. *94*:89-110, 1931.

104. Beard, H. H., R. W. Baker, and V. C. Myers. Studies in the Nutritional Anemias of the Rat; V. The Action of Iron and Iron Supplemented with Other Elements Upon the Daily Reticulocyte, Erythrocyte, and Hemoglobin Response. J. Biol. Chem. *94*:123-134, 1931.

105. Barannik, P. I., I. A. Mikhalyuk, I. N. Motuzkov. Data on the Role of Chromium, Lead, Vanadium and the Natural Radioactivity of Foodstuffs in the Etiology of Endemic Goiter. Gig. Sanit. (Moscow) English Ed., *35*(1-3):289-291, January-March. 1970.

106. Selyankina, K. P. Data for Determining the Maximum Permissible Content of Vanadium in Water Basins. Gig. Sanit. (Moscow) *26*(10):6-12, Oct. 1961.

107. Cannon, H. L. The Biogeochemistry of Vanadium. Soil Sci. *96*:196-204, 1963.

108. Allaway, W. H. Agronomic Controls over the Environmental Cycling of Trace Elements. Advan. Agron. *20*:235-274, 1968.

109. Pratt, P. F. Vanadium. *In:* Diagnostic Criteria for Plants and Soils. Riverside, Calif.; University of California Press. 1966, p. 480-483.

110. Warington, K. Interaction between Iron and Molybdenum or Vanadium in Nutrient Solutions With or Without a Growing Plant. Ann. Appl. Biol. *44*:535-546, *1956*.

111. Prince, A. L. Trace Element Delivering Capacity of 10 New Jersey Soil Types as Measured by Spectrographic Analysis of Soils and Mature Corn Leaves. Soil Sci. *84*:413-418, 1957.

112. Stanier, R. Y., M. Doudoroff, and E. A. Adelberg. The Microbial World. Englewood Cliffs, Prentice-Hall, Inc., 1970. p. 233.

113. Burk, D., and C. K. Horner. The Specific Catalytic Role of Molybdenum and Vanadium in Nitrogen Fixation and Amide Utilization by *Azotobacter*. Trans. Int. Congr. Soil Sci. 3rd Congr. Oxford *1*:152-155, 1935.

114. Horner, C. K., D. Burk, F. E. Allison, and M. S. Sherman. Nitrogen Fixation by *Azotobacter* as Influenced by Molybdenum and Vanadium. J. Agr. Res. *65*:173-193, 1942.

115. Zajic, J. E. Microbial Biogeochemistry. New York, Academic Press, 1969. p. 196-202.

116. Nicholas, D. J. D. Role of Trace Metals in the Nitrogen Metabolism of Plants, with Special Reference to Microorganisms. J. Sci. Food Agr. (suppl. issue). *8*:15-25, 1957.

117. Bertrand, D. Survey of Contemporary Knowledge of Biogeochemistry. 2. The Biogeochemistry of Vanadium. Translated by Vera Lee. Bull. Amer. Museum Nat. Hist. *94*:407-455, 1950.

118. Arnon, D. I. and G. Wessel. Vanadium as an Essential Element for Green Plants. Nature. (London) *172*:1039-1040, 1953.

119. Alexopoulos, C. J. Introductory Mycology (2nd Ed.). New York, John Wiley and Sons, 1962. p. 518.

120. The Merck Index (8th Ed.). Rahway, N. J., Merck and Company, 1968.

121. Romoser, G. L., W. A. Dudley, L. J. Machlin, and L. Loveless. Toxicity of Vanadium and Chromium for the Growing Chick. Poultry Sci. *40*:1171-1173, 1961.

122. Berg, L. R. Evidence of Vanadium Toxicity Resulting from the Use of Commercial Phosphorus Supplement in Chick Rations. Poultry Sci. *42*(3):766-769, 1963.

123. Hathcock, J. N., C. H. Hill, and G. Matrone. Vanadium Toxicity and Distribution in Chicks and Rats. J. Nutr. *82*:106-110, 1964.

124. Schroeder, H. A. and J. J. Balassa. Arsenic, Germanium, Tin and Vanadium in Mice: Effects on Growth, Survival and Tissue Levels. J. Nutr. *92*:245-252, 1967.

125. Schwartz, K. and D. B. Milne. Growth Effects of Vanadium in the Rat. Science. *174*:426-428, 1971.

126. ter Heege, J. H. Een Intoxicatie bij Runderen Door Opname van Stookolieroet (Poisoning of Cattle by Ingestion of Fuel Oil Soot). Tijdschr. Diergeneesk. *89*:1300-1304, 1964.

127. Additives Cut Pollution when Firing Residual Oils. Mod. Power Eng. *65*(6):70-71, 1971.

128. Exley, L. M., A. E. Tamburrino, and A. J. O'Neal. LILCO Trims Residual Oil Problems. Power. *110*(4): 69, April 1966.

129. Belyea, A. R. Manganese Additive Reduces SO_3. Power. *110*(11):80, Nov. 1966.

130. Papamarcos, J. Fuel Oil Additive Passes Test. Power Eng. *75*(4):46-48, April 1971.

131. Reid, W. T. External Corrosion Deposits: Boilers and Gas Turbines. New York, American Elsevier, 1971. p. 134-188.

132. Thompson, R. N., J. E. Zajie, and E. Lichti. Spectrographic Analysis of Air-dried Sewage Sludge. J. Water Pollut. Contr. Fed. *36*(6):752-759, June 1964.

133. Berrow, M. L., and J. Webber. Trace Elements in Sewage Sludges. J. Sci. Food Agr. *23*(1):93-100, Jan. 1972.

134. Gross, M. G. Analyses of Dredged Wastes, Fly Ash, and Waste Chemicals — New York Metropolitan Region. Marine Sciences Research Center. State Univ., Stony Brook, New York. Tech. Rept. No. 7. DDC NTIS AD 734337, October 1970.

135. Bowen, H. J. M. Trace Elements in Biochemistry. London, Academic Press, 1966.

136. Jaworowski, Z., et al. Radiation to Population from Conventional and Nuclear Power Production. *In*: Symposium on Environmental Surveillance Around Nnclear Installations. International Atomic Energy Agency, Warsaw, IAEA-SM-180/20, 1973.

7. CONTROL TECHNOLOGY

Control strategy for vanadium emissions must be based on effective removal of particulate material from process exhaust streams. Technology exists for providing extremely good control of particulate emissions from industrial processes and combustion sources. The degree to which control of vanadium emissions is achieved will be proportional to the extent of application of high-efficiency particulate control devices.[1]

Many industries have applied effective control devices to their process stream. For example, Lee et al.[2] have reported total removal of vanadium content of a steel mill process stream by a baghouse. Although no data are available specifically for vanadium, high total mass collection efficiency has been attained on metallurgical exhaust streams using electrostatic precipitators and wet scrubbers as well as baghouses.[3,4]

Coal-fired power generating facilities have used electrostatic precipitators (ESPs) for a number of years. Recent years have seen the installation of ESPs capable of removing more than 99.5 percent of the particulate loading. Lee et al.[2] reported on a precipitator that removes 99.85 percent of the vanadium in the exhaust stream. Some of the input vanadium is captured in the bottom slag and open boiler surfaces, so that total removal is even better than this figure. Lee's data show that most of the escaping vanadium is below 5 μm in diameter, and almost half is less than 1 μm. McCain[5] has reported removal efficiencies above 90 percent for particles as small as 0.06 μm by ESPs on coal-fired boilers. A baghouse has proved effective on an anthracite coal-burning unit with total collection efficiency greater than 99.5 percent. More effective control of vanadium from utility coal-fired boilers can be expected as more high-efficiency units are installed to meet increasingly stringent local emission laws.

Particulate control of oil-fired combustion sources has been practically nonexistent because total mass emissions are usually less than local regulations require. Vanadium recovery has been reported as a result of attempts to control boiler corrosion by means of magnesia-alumina fuel additive.[6] Enough vanadium has been recovered to make the process economically attractive, with 75 percent of the input vanadium recoverable from the bottom slag. Control of the particulate emissions may be enhanced if sulfur dioxide removal schemes such as the Chemico magnesium oxide process are applied to oil-fired combustion sources. High removals of particulate have been reported with this system.[7]

The enormous volume of vanadium-containing fuel consumed means vast quantities will still be emitted until even better devices are available. A high degree of control of vanadium may be expected from stationary sources as new particulate or sulfur dioxide control devices are extended to all possible sources.

7.1 REFERENCES FOR SECTION 7

1. Vanadium (Section V). *In:* National Inventory of Sources and Emissions: Arsenic, Beryllium, Manganese, Mercury and Vanadium Emissions. W. E. Davis and Associates, Leawood, Kansas, 1971.

2. Lee, R. E., Jr., H. L. Crisp, A. E. Riley, and K. MacLeod. The Concentration and Size of Trace Metal Emissions from a Power Plant, a Steel Plant, and a Cotton Gin. Environ. Sci. Technol. (In press).

3. Harris, D. B. and D. C. Drehmel. Fractional Efficiency of Metal Fume Control as Determined by Brink Impactor. U. S. Environmental Protection Agency, National Environmental Research Center, Control Systems Laboratory, Research Triangle Park, N. C. (Presented at 66th Annual Meeting of the Air Pollution Control Association, Chicago, June 24-28, 1973.)

4. Patchell, G., and R. N. Allen. Electric Furnace Testing — Particle Size Measurements. Resources Research, Inc. Reston, Va. EPA Contract CPA 70-81, March 1972.

5. McCain, J. E. Particle Sizing Techniques for Control Device Evaluation. Southern Research Institute, Birmingham, Ala. EPA Contract 68-02-0273, October 1973.

6. Lee, G. R., F. D. Friedrick, and F. D. Mitchell. Control of SO_3 in Low Pressure Heating Boilers by an Additive. J. Inst. Fuel. *42*:67, 1961.

7. Statnick, R. M. U. S. Environmental Protection Agency, National Environmental Research Center, Control Systems Laboratory, Research Triangle Park, N. C. Personal communication with Chemico Corp. 1973.